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SYMPOSIUM ON NUTRITION AND THE DEFICIENCY DISEASES

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HARVARD AND NUTRITION*

CHAIRMAN'S ADDRESS

BY GEORGE R. MINOT, M.D.†

AMONG John Harvard's books was a copy of "Directions for Health," by Sir William Vaughan that passed through many editions. This treatise gives directions as to diet, so that the Harvard authorities may well have been acquainted with this problem from the early days of the college. Certainly, within the second academic year, they must have sensed the importance of proper food for their students. Although the wife of the first "professor", Nathaniel Eaton, was responsible for the students' fare, the magistrates of the court learned she often gave them only porridge and pudding, never beef, and the bread was sometimes "made of heated sour meal". Harvard's first "dietetian", however, branded as calumny the further assertion that their mackerel were cooked ungutted and that there was "goat's dung in their hasty pudding". It is still true today that good food may be spoiled by a poor cook. This state of affairs was soon corrected, but in those early days "short commons" were heard of from time to time. Indeed Nathaniel Mather, writing from England, asked, "Hath the meagrenesse of their winter commons shrunk up their guts and made their braines to perish, that they have quite forgotten us?" Although such remarks may suggest the occurrence of dietary deficiency disease, there is ample evidence that by and large the colonists had good food. Edward Johnson, in his "Wonder Working Providence", wrote in 1654, "The poor wilderness hath not onely equalized England in food but goes beyond in some places."

From antiquity until modern days, disease was considered largely due to some positive injurious agent. Concepts of diet chiefly concerned what foods were harmful. To diet meant "to be deprived in large measure of the pleasures of the table". Today we know that the lack of substances is an important cause for ill health. There are, of course, harmful effects from excess of food; but the underfed and misfed, owing to a deficit in one or more aspect of the diet, create far more important problems. The major problems of nutrition concern supplying a diet optimal in multiple factors for each given individual at each stage of life. The energy requirements of the body must be met as well as many other specific requirements. The addition of vitamins and minerals does not replace calories. Quality, although of fundamental importance, cannot be substituted for quantity. The desire to be a slim young lady may lead to emaciation but not necessarily a dietary deficiency disease.

The science of the physiology of metabolism was founded by Sanctorius, who died the year Harvard College was founded. The names of Lavoisier, von Voit, Rubner, Atwater and Lusk are among many others intimately associated with the development of knowledge of metabolism and nutrition. Studies on nutrition at Harvard were few during the 150 years when these men made fundamental observations. J. C. Warren, James Jackson and Jacob Bigelow recognized the importance of diet for health, and Warren has been considered to have "made a science of the regulation of food". But the emphasis of these men was on overeating, although Jackson gave consideration to the quality of food. Henry P. Bowditch first established the correct curve for growth of man, and this was further studied by W. T. Porter. These studies and the extensive ones by Charles S. Minot on growth have a bearing on problems of nutrition. Rotch's contributions to infant nutrition, begun in 1887, were significant. He gave birth to the idea of individualization and variation and looked upon nutritional disturbances as being caused by certain elements of the diet rather than by diet as a whole. Rotch emphasized the necessity of thinking of food in terms of its components. Frederick C. Shattuck was one of the leaders in the recognition that the increased caloric requirements of typhoid fever patients must be met. A new wave

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of interest in metabolism began about 1908 when Benedict, at the Carnegie Nutrition Laboratory, described his new "unit respiration" apparatus. His work and that at Cornell of DuBois, a graduate of Harvard College, by means of clinical calorimetry have led to important advances. Joslin, at first with Benedict, has made extensive metabolic studies of diabetes, and Talbot, originally working with Benedict, has formulated the energy requirements of infants and children. The metabolic studies at Harvard concerning endocrine disorders are outstanding, being led by Means and Aub under the original stimulus of Edsall. Boothby of the Mayo Clinic also began his metabolic studies while working at the Peter Bent Brigham Hospital. During this twentieth century the contributions of Otto Folin to nitrogen metabolism and to the development of biochemical methods have done much to aid in the understanding of nutrition problems.

About 25 years ago was the dawn of the "newer knowledge of nutrition", when it became recognized that for health, in addition to protein, a source of energy in the form of protein, carbohydrate or fat, and a suitable supply of certain organic salts, it was also necessary to have a particular quality of protein and what are now called vitamins. More has been learned about the science of nutrition in the past quarter century than in any other period of the world's history. *Nutrition Abstracts* lists for the past year about 5000 articles on the subject. The development of knowledge has, however, not been entirely beneficial in its application, which is certainly unfortunate considering that about fifteen billion dollars a year are spent for food in the United States alone. The public's enthusiasm for vitamins and minerals illustrates well that "a little learning is a dangerous thing". No advantage to the public health has been gained by clever advertising emphasis on the infinitesimal amounts in which vitamins occur in ordinary foodstuffs and the exploitation of the "vitamin conscious" portion of the population with costly proprietary foods and remedies of uncertain vitamin content. The popularization of the relatively recent and constantly changing knowledge has also resulted in very little intelligent understanding of the facts.

In recent years there has been very significant work at Harvard concerning some of the modern aspects of nutrition, and you will hear shortly from some of the contributors themselves. Harvard was rather late in actively entering the vitamin field, and such names as Eijkman, Hopkins, Funk, McCollum, McCarrison and Mendel are not found on her roster. Yet Oliver Wendell Holmes in 1861 said, "I cannot help believing that medical curative treatment will by and by resolve itself in a good measure into modifications of food swallowed. . . . The effects of . . . cod liver oil . . . are only hints of what will be accomplished when we have learned to discover what organic elements are deficient or in excess in a case of chronic disease." Today the University has no man trained throughout life as a student of nutrition. There are here, however, diverse activities and potential opportunities to serve as a background for the further development of such studies which can have a far-reaching influence in many different fields. The mutual interaction of the laboratory and the ward will aid to solve problems of nutrition in relation to disease.

Nutrition intimately concerns the welfare of man, and his place in future history will depend in no small part on what he decides to eat. If the optimal, not usual, diet for man at all ages and under varying circumstances of his activity and environment were known and if, throughout generations, each person took an ideal diet—one nicely adjusted with respect to all its constituents at an optimum level for the best possible achievement—not only would much illness be prevented but the physical and mental development of man would be improved, leading to consequences of vast importance. Nutritional studies can wisely concern every phase of metabolism of organisms and their component cells. The effects of unsatisfactory nutrition play important roles in economic, educational, and sociologic problems, as well as induce disorders referable to every branch of medicine and dentistry. The effects of food on vitality, normal development, reproduction and longevity are fundamental and widespread, and there are multiple opportunities throughout the world for prevention of many sorts of disorders and of some unsocial states by better nutrition.

In the United States the essential food elements are abundant and easily procured. Individual low purchasing power plays some role in the production of faulty nutrition, but more often it is due to ignorance of the principles of good nutrition or to faddism. The physician must be scrupulously careful in prescribing diets, for example for diabetes or peptic ulcer, not to allow them to be deficient in any respect. Dietary deficiency may arise not only because of an improper intake of one or more substances required by the body, but also because established disease may prevent the formation, absorption and utilization or cause abnormal loss from the body of necessary substances. The effects of extra demands are always to be reckoned with. The physiologic strain of childbearing requires dietary factors to be from 10 to over 100 per cent greater than the standard requirements for women. Moreover, since the maternal diet in pregnancy and lactation inevitably affects the well-being of the infant, the

health of the whole population depends to a greater or less extent on the nutrition of the mothers. With increased metabolism from exercise or other causes, the demands for various essential substances such as vitamins B and C increase, while the requirement of minerals and vitamins is far greater during growth than in adult life. The influence of infection in decreasing the effectiveness of liver extract, iron, vitamin C and the like is now well recognized. The major problems of nutrition do not concern clear-cut deficiency diseases but the prevention of the occurrence of partial deficiency. Borderline states of nutritional instability are much more common than is usually appreciated. There is a wide zone between optimal nutrition and the level at which classic symptoms of recognized dietary deficient states develop. The undesirable influence of a faulty diet in the zone of partial deficiency may become detectable only after years or generations. Departure from an optimal diet, even if slight, may produce illness when operative over a long period of time, especially if combined with digestive disturbances or conditions inhibiting the utilization of substances derived from food. Such adverse factors as infection, chronic fatigue and arteriosclerosis can act to precipitate distinct dietary deficiency syndromes when a state of nutritional instability exists. Rapid improvement follows adequate treatment of deficiency disease but quick results in the health and longevity of a people are not to be expected through better diet.

The determination of the existence of a mild state of suboptimal nutrition is difficult. A carefully taken diet history may be of great value. Signs of improper development, especially of the skeleton, or anemia may be present. Symptoms may be absent or only vague, such as a lack of sense of well-being. Rapidly increasing knowledge such as that concerning the chemical constitution of some of the vitamins is leading to procedures that aid us to determine if a deficiency exists.

The principle of treatment of deficient states consists in supplying the deficient substances on a quantitative basis. The outstanding signs of deficiency usually rapidly vanish when enough material enters the body to place the individual high in the zone of partial deficiency. The deficient state must be met in toto; for example, in pernicious anemia a certain amount of liver or stomach preparation is needed to maintain a fairly satisfactory condition with over four million red blood cells, but it takes much more to meet all the individual's requirements. Today's knowledge does not permit us to prescribe with precision the amounts of the thirty-six or more substances which are required for correct nutrition. We must be guided by the general principles derived from the results of research and practical experience. To detect deficiencies and to remedy them piecemeal by supplements of manufactured concentrates will not at present solve the problem. Experience tells us that a mixed diet of natural food stuffs, one especially rich in milk, green vegetables, fruit, butter, eggs and food with ample protein of good biologic value gives the best results.

As one looks back to the time of the celebration of the two hundredth year of this University, we may say that essentially nothing was known about nutrition. We should probably shudder if we knew what our successors will say one hundred years hence about our meager knowledge of that subject. Yet we may take satisfaction in knowing that the foundations for their knowledge have been laid. It remains for us to seek diligently for more information in the laboratory and at the bedside. We may thus carry forward the work that the ultimate goal may be nearer for those who follow us.

CHAIRMAN MINOT: I have made no allusion to one very essential dietary constituent, namely water. Its physiologic success has been critically considered by Professor Lawrence J. Henderson. Any marked departure from the normal content of body water gives rise to consequences which may prove serious. The minimal normal requirement for adult man is about 1500 gm. every twenty-four hours, but this may be far too little to supply the demands in certain pathologic states. Upon the distribution of water in the body depends the translocation of other materials and, to a certain extent, this relationship is reciprocal. Thus, upon the content of water in each tissue depends the continuation of the activity of that tissue. It is often convenient to look upon water of the body as occurring in several compartments. They are distinguished not only by location, but by the particular electrolytes in solution. The major portion of body water is in the cells. The extracellular body fluids, one-quarter of which are in blood plasma, form about 20 per cent of the body weight. These fluids bring nutrient material to the tissue cells and carry away waste products of cell activity. They also provide a stability of certain physiochemical conditions within the organism which are fundamental for life. There are few men in this world as well fitted as Dr. James L. Gamble, Professor of Pediatrics, to discuss the importance of the "Extracellular Fluid and Its Maintenance" to our life and happiness:—

EXTRACELLULAR FLUID AND ITS MAINTENANCE*

BY JAMES L. GAMBLE, M.D.†

THE extracellular body fluids are the plasma of the blood and the interstitial fluid (including lymph) that lies between the vascular compartment and the tissue cells, as roughly shown by the diagram in figure 1. Extracellular fluid constitutes, as Claude Bernard appreciated, the immediate environment of the organism. This aqueous medium which surrounds the tissue cells sustains two important services. In the first place it conveys nutrient and waste materials. With this transport function we have long been familiar. The other service which extracellular fluid must provide, our knowledge of which we owe largely to Lawrence Henderson, is a stability of physicochemical conditions within the organism. Prominent among these are reaction in terms of hydrogen ion concentration, osmotic pressure and temperature. The values for these physical properties in cell fluid rest on the values at which they are held in the surrounding medium. The necessity for nearly stationary values may be indicated by recalling that the rates at which processes of chemical change proceed are, in widely varying degree, affected by alterations in the accompanying physical circumstances.

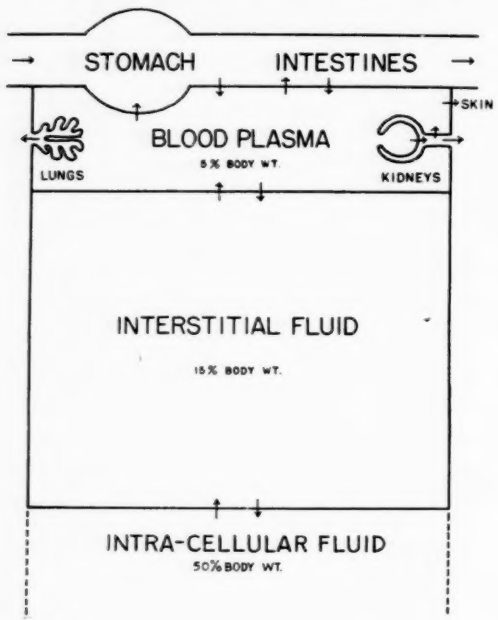

FIGURE 1.
International Clinics, Vol. 2, Series 46.
Philadelphia: J. B. Lippincott Co., 1936.

It is therefore evident that the innumerable and interrelated chemical reactions that together accomplish what we call metabolism would rapidly fall out of adjustment, if these underlying conditions were not held at fairly constant values.

The chemical anatomy of extracellular fluid is described by the two middle diagrams in figure 2. In these diagrams the relative values for the concentrations of the chemical components of extracellular fluid are shown. The values for the cations or potential base are superimposed in the left hand columns and those for the anions or acid radicals in the right hand columns. The values for the nonelectrolytes, that is substances which do not dissociate into anions and cations, are shown across the diagram. Except for the very much larger concentration of protein in the blood plasma, the chemical structure of the two extracellular fluids is, as may be seen in the diagrams, almost identical.

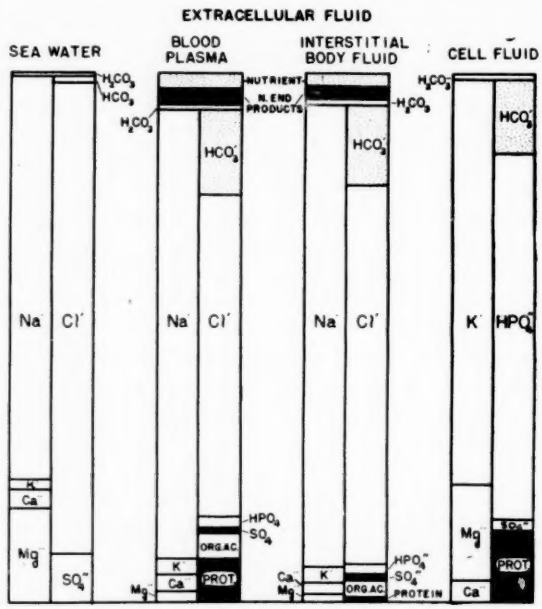

FIGURE 2.
International Clinics, Vol. 2, Series 46.
Philadelphia: J. B. Lippincott Co., 1936.

As regards the history of extracellular fluid, the first diagram describes the chemical composition of the aqueous medium in which the earliest forms of life were successful. The resemblance of the electrolyte pattern of sea water to that of extracellular fluid is very striking. We find the same four components of the total base value, the same prominence of the concentrations of sodium and of chloride ion, and the same pair of buffering substances, free car-

*An abbreviated presentation of material used in the following article by the author: Extracellular Body Fluid. International Clinics, Vol. 2, Series 46. Philadelphia: J. B. Lippincott Co., 1936.

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bonic acid and bicarbonate. In other words the chemical skeleton of sea water is clearly visible in extracellular fluid.

That extracellular fluid is a surrounding and not a pervading medium is shown by the widely different electrolyte pattern of cell fluid which is described by the last diagram in figure 2. Quite recently it has become fairly well established that the two most prominent factors of the chemical structure of extracellular fluid, sodium and chloride ion, are not permitted to pass the boundaries of protoplasm. That is, their position is exclusively extracellular. Obviously this circumstance should facilitate accuracy of control of the quantities of sodium and chloride ion in extracellular fluid, a matter of importance since, as will presently appear, these two factors constitute a framework on the stability of which the necessary constancy of the physicochemical properties of extracellular fluid almost entirely depends. To turn for a moment to the concentration of bicarbonate ion (HCO_3) which is a prominent part of the mechanism that defends body fluid reaction, this concentration of bicarbonate ion is constructed from that largest end product of metabolism, carbonic acid, to such extent as the concentration of total base stands above that of the other acid radicals. Owing to this mendicant position of the bicarbonate concentration, defects developing in other parts of the electrolyte structure cause a change in the bicarbonate value. This is illustrated by the diagrams in figure 3. In the second diagram the concentration of the other acid radicals (A) is above its usual value with the result that the quantity of base available for the covering of bicarbonate ion is correspondingly reduced. In the next diagram is shown the reverse change which produces an extension of bicarbonate. In the last diagram a reduction of the total base value is seen to cause an equal reduction of the concentration of bicarbonate ion. Referring again to figure 2, it may be seen that the normal value for the bicarbonate ion concentration depends chiefly on an accurate control of the two large structural factors, sodium and chloride ion.

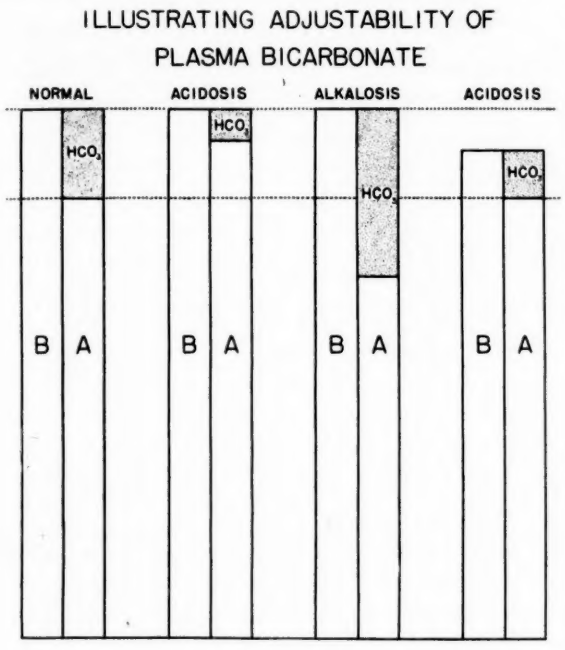

FIGURE 3.
International Clinics, Vol. 2, Series 46.
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The osmotic value of extracellular fluid is, of course, determined by the sum of the concentrations of all of its chemical components. Here the concentration of sodium, owing to its large size and also to the adjustability of the bicarbonate ion concentration, plays an almost determining role. That is, the adjustable factor being on the acid side it follows that the total base value determines the height of the electrolyte structure or, in other words, the osmotic value of the electrolytes. The contribution of the nonelectrolytes to the total osmotic value is, as may be seen in the diagram, relatively small, and it is also seen that nearly all of the total base is sodium. We thus find that the osmotic value of extracellular fluid rests almost entirely on control of the concentration of sodium. Chloride ion, the other large skeletal factor, plays no direct part in determining the osmotic value because of the circumstance that a change in the concentration of chloride will be offset by a reciprocal change in the concentration of bicarbonate ion. We can readily see, however, that the concentration of chloride ion, by filling in the space between a suitable concentration of bicarbonate ion and the other relatively very small acid values, permits the concentration of sodium to a magnitude that enables it practically to determine the osmotic value.

We thus find surrounding the tissue cells a fluid of quite remarkable chemical ingenuity. Obviously this fluid is constructed from materials derived from the water and food intake. As regards control of these materials, except for the concentration of free carbonic acid which is held at a fixed value by the respiratory mechanism and the concentration of protein which is established by a mechanism which is not yet visible, the chemical pattern of extracellular fluid is sustained by the kidney. The kidney is very inadequately described as an organ of excretion. Were the removal of waste products its only function, a much simpler organ would suffice. Its complexity of design and intricacy of function are required for the construction and accurate defense of extracellular fluid on the chemical constancy of which depends the successful operation of vital processes. Macal-

lum, regarding the establishment of the enclosed aqueous medium as the largest forward step in the history of the animal organism, has described the kidney as the organ *par excellence* of evolution.

For the maintenance of extracellular fluid only three materials are directly required: water, sodium and chloride ion. All of the other items of its chemical structure are provided by the processes of cell activity. This is not the case for sodium and chloride ion because of their extracellular position. Nor is the water of oxidation released by metabolic processes nearly sufficient to cover the expenditure of water in the removal of waste products in urine and in the dissipation of heat from the body by the vaporization of water. We can therefore understand why two special sensations were devised to insure an adequate maintenance of extracellular fluid—the sensation of thirst and a specific craving for sodium chloride. These considerations also enable us to understand the remarkable efficacy of an extremely simple therapeutic agent, physiologic salt solution, for repair of defects in the volume and structure of extracellular fluid.

CHAIRMAN MINOT: Alterations in the extracellular body fluids may lead to edema. Decrease of colloid osmotic pressure exerted by the plasma proteins is an important factor in the accumulation of fluid in the tissues and serous sacs. The commonest method of depriving the body of protein is through a prolonged or excessive reduction of protein in the diet. In 1860, Bischoff and Voit noted that a bread diet led to a negative nitrogen balance and an increase in body water. It has been appreciated only recently, however, that there is a close connection between protein deficiency and edema, although the occurrence of dropsy with undernutrition has attracted attention for centuries. Like other deficiency diseases, nutritional edema has appeared in epidemic form under conditions which impose severe odds in man's struggle for existence, and it has been described under such names as epidemic dropsy, war, prison or famine edema and hunger swelling. Protein deficiency may inhibit growth and also cause symptoms, such as muscle weakness, without the appearance of visible edema. Nutritional edema in a pure and marked form is rare but mild degrees are common in the course of other illnesses. Although nitrogen starvation is of prime importance, there are many accessory factors which must be taken into consideration. This has been demonstrated in connection with edema following surgical operations, especially by Dr. Chester M. Jones at the Massachusetts General Hospital, who will now discuss "Protein Deficiency":

PROTEIN DEFICIENCY

BY CHESTER M. JONES, M.D.*

DR. Minot has commented upon at least one of the causes of protein deficiency. He has also suggested that this deficiency, like most others, is to be observed only rarely in what might be called a pure state. I should like to amplify his remarks and to comment upon the symptoms due to protein depletion, the mechanism underlying them and their treatment.

An exact definition of protein deficiency is difficult, but it is probably best described as one in which the deficiency is reflected in a lack of available protein in the plasma with a tendency to edema and serous effusions. There are several important causes — an insufficient protein intake, inadequate absorption of protein, an abnormally great loss of protein from the body, increased destruction of protein in the body and insufficient regeneration of protein. Protein depletion may develop acutely, but as a rule its manifestations are to be considered as the result of a chronic condition.

The simplest and the most obvious cause of protein lack is an insufficient intake.

In ordinary civil life there are two common causes. Extremely bad economic conditions in individual families may result in a limitation of meat and other nitrogenous food. A second cause, and one that is extremely important, is the presence of an underlying local or constitutional disease with marked anorexia resulting in a limitation of dietary protein. This is true in diseases of the digestive tract, especially if there is any degree of obstruction with vomiting, and is of particular importance inasmuch as the resulting food and protein lack may seriously complicate necessary surgical measures. In other words, the deficiency secondary to the fundamental condition may actually determine the success with which the underlying disease is handled.

Diseases such as diabetes, because of the inadequate utilization of food, are in a sense equivalent to a deficient food intake and are not infrequently associated with a protein deficiency, which, however, is almost always a part of a general dietary lack.

A somewhat less common cause is that of protein loss, which may be due to several pathologic conditions. Massive hemorrhage may provide an acute loss of plasma protein which the organism is unable to regenerate before the development of clinical symptoms. Obviously the hemorrhage must be of major proportions, and in addition there must be present an associated chronic disease that is the cause of the bleeding and that in itself retards the recovery from the sudden loss of plasma. Such an event may occur during the course of a bleeding ulcer

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or in cirrhosis of the liver with bleeding from esophageal varices. Under such conditions massive hemorrhage may precipitate acute protein deficiency with its resulting clinical symptoms.

Chronic protein loss occurs typically in the nephrotic syndrome with its striking albuminuria. In extreme cases the plasma protein may reach a very low figure. To a lesser degree chronic protein lack may result from the other forms of renal insufficiency because of the associated albuminuria. Less common is the depletion seen in the later stages of cirrhosis. It is possible that in this type of liver disease, as in many other degenerative conditions, there is a failure to form protein properly, but in the later stages of cirrhosis there is an appreciable loss of protein in the ascitic fluid. When the ascites recurs at a rapid rate, so that frequent taps are necessary, the loss of albumin may reach proportions that are of real importance. A somewhat analogous situation occurs in the rather rare condition of adherent mediastinopericarditis, with its mediastinal and portal venous stasis and resultant serous effusions. Obviously any chronic exudative disease that necessitates frequent tapping or drainage may serve as the basis for protein depletion.

Failure of absorption of protein can occur under varying circumstances. Obviously, long-continued diarrhea, due to such diseases as ulcerative colitis, tuberculous enteritis and the like, may result in a striking failure of absorption with consequent protein lack and low plasma protein values. Malignant disease of the lower intestinal tract occasionally may produce a diarrhea of sufficient severity to bring about a similar result. In sprue, pernicious anemia, beriberi, and so forth, absorptive processes may be inadequate because of the increased intestinal rate and also because of the changes in the intestinal mucosa. During relapses in these deficiency states it is becoming well recognized that striking mucosal alteration occurs which can seriously interfere with secretory and absorptive processes. Thus the immediate results of a specific deficiency state, such as pernicious anemia, may be the development of a further specific type of deficiency due to a malfunctioning digestive tract.

Abnormal protein destruction and diminished protein regeneration are undoubtedly operative in many cases of protein depletion, but such factors are difficult to evaluate. Obviously, in severe febrile states, in which the processes of catabolism are increased and the food intake is decreased, minor degrees of protein lack may develop. With more chronic febrile disturbances, such as occur in subacute bacterial endocarditis, pulmonary tuberculosis and the lymphomas, there may well be a sufficient destruction of body protein to cause a real protein deficiency with its resultant clinical manifestations. In such instances the factor of increased destruction is almost always coupled with an inadequate intake.

The regeneration of body protein is not at all clearly understood. Fibrinogen is known to be formed by the liver, but this protein constitutes only 3 to 6 per cent of the total plasma proteins, and at present one can only speculate as to the origin of the other albumin and globulin components. It is probable that in the chronic degenerative diseases the processes concerned with the normal regeneration of body and plasma protein are interfered with sufficiently to contribute to a definite protein deficiency.

In the presence of serious protein lack certain clinical symptoms occur. Muscle wasting, with its associated weakness, is the obvious result of the loss of body protein from any of the causes already discussed. The lowering of the plasma protein is the direct result of the protein lack. It has been well established that the albumin fraction of the blood is the most rapidly depleted and the most slowly regenerated in cases of protein deficiency. Because the molecular size of the albumin components of the plasma proteins is definitely much less than that of the globulins, any striking diminution of the albumin content of the plasma results in a marked lowering of the osmotic pressure in the circulating blood, with an associated tendency to edema and serous effusion.

This tendency to form abnormal accumulations of water in the tissue spaces or in the serous cavities is characteristic of protein deficiency regardless of the underlying cause. As a rule the edema is subcutaneous and is to be noted only in the extremities. As such it is chiefly of importance as an indication of the severity of the underlying condition. Too frequently it is assumed that it is due to cardiac or renal failure, and the true cause is overlooked. In addition to the peripheral edema one may observe somewhat less commonly an accumulation of fluid in the serous cavities. This may also occur in the absence of demonstrable edema. Serous effusions due to protein lack are usually to be noted in cases of nephrosis, cirrhosis of the liver, mediastinopericarditis and the like. A third location for the abnormal deposition of fluid exists, but this is not commonly recognized. This is in the viscera themselves. Edema of the lungs is commonly attributed to cardiac failure. That it may occur as the result of protein lack or that such viscera as the heart or the intestine may also participate in an abnormal accumulation of water is less well known.

A few years ago experimental observations provided us with some very interesting data concerning such a phenomenon. In our experiments animals were subjected to varying degrees of pure protein starvation. Once the plasma protein

level had been definitely reduced, various maneuvers were carried out, such as the establishment of sterile drainage from the peritoneal cavity, the production of sterile subcutaneous abscesses and the performance of an enteroanastomosis. At the same time adequate or slightly excessive amounts of fluid in the form of normal salt solution were administered subcutaneously. By such means various postoperative situations were closely simulated. After varying periods of time, which were more or less dependent on the degree of protein starvation preceding the operative procedure, peripheral edema usually developed. Free fluid was noted frequently in the serous cavities. To us, however, one of the most interesting findings was the very striking increase in the water content of the viscera. We found as much as 10 per cent increase in the water content of the lungs, 5 per cent in the heart muscle and 7 per cent in the wall of the ileum. Such increases in the water content of the organs imply a marked change in tissue physiology, and it is hard to believe that such changes are consistent with normal function. It seems equally logical to suggest that such variations from the normal may frequently constitute the background for the so-called "poor-risk" patient.

The development of nutritional edema is not dependent solely upon the actual level of the plasma protein or albumin. Even with extremely low values edema will not occur without a sufficient fluid intake. Limitation of fluid, due to an insufficient intake by mouth or other routes or by reason of excessive vomiting or diarrhea, may definitely influence the course of abnormal water accumulation in the body. In cases of advanced nephritis striking edema has been known to disappear during the period of vomiting associated with the development of uremia. In spite of a very low plasma protein, it was not possible to produce edema in our experiments, even in those animals in which depletion was extreme, unless the amount of fluid administered was somewhat in excess of the normal daily intake. Another variable of equal importance is the intake of sodium chloride. Limitation of this substance prevented or diminished the abnormal deposition of water in the tissues. The functional condition of the heart, kidneys and liver, the presence of sepsis and increased capillary permeability are all additional factors of importance, but, without question, the lowering of the plasma protein and albumin values constitutes the most important single factor affecting the development of the outstanding symptom of edema in cases of protein deficiency.

A brief presentation of two cases will best illustrate the phenomenon under discussion and will indicate how the condition may develop under circumstances other than those of simple starvation.

The first case is that of a 62 year old man with a typical story of partially obstructing pyloric ulcer of fifteen years' duration. He entered the hospital because of vomiting. Examination showed loss of weight and edema of the legs, genitals and abdominal wall. The plasma protein was 5.7 per cent. With rest and careful feeding the edema disappeared, and a gastroenterostomy was performed. During the first week after the operation vomiting recurred, and at the end of this period edema developed rapidly. Oliguria was an associated finding, with the specific gravity of the urine reaching 1.034. On the tenth day a watery diarrhea developed, suggesting edema of the intestine, and two days later pulmonary edema was noted. He died the following day. The plasma protein one week after the operation was 4.5 per cent. Necropsy showed gross edema and fluid in the serous cavities. The malfunctioning of the gastroenterostomy was due to a striking edema of the walls of the jejunum and ileum, with partial obstruction of the site of operation due to the swelling.

This case is typical of protein deficiency due to the malnutrition accompanying gastrointestinal disease. The necessary surgical measures could but aggravate the existing protein lack, and the required fluid and salt that had to be administered postoperatively could hardly fail to cause edema with the existing low plasma protein. An almost unavoidable vicious circle was established, with the rapidly developing visceral edema completing the final picture of tissue failure.

The second case is an example of acute protein depletion. The patient, a man of 40 years, entered the hospital because of acute hemorrhage. There was a history of chronic alcoholism, with recent symptoms of anorexia and mild indigestion. Examination showed marked pallor, sweating and a rapid pulse. Light percussion revealed a definite increase in splenic dulness. The hemorrhage was of sufficient degree to give a red blood cell count of only 1,500,000 and a serum protein of 4.5 per cent. Subsequent x-ray examination showed esophageal varices. The particular point of interest was the sudden development of ascites and edema about a week after the bleeding had subsided. This was undoubtedly due to an acute protein deficiency, caused by the hemorrhage and aggravated by three other factors—the inability to take sufficient protein by mouth, the necessity of administering moderately large amounts of fluid by clysis and the inability of the liver to function normally in relation to the regeneration of plasma protein. With the gradual return of the blood to a more normal level the ascites and edema disappeared.

The treatment of protein deficiency depends upon the factors causing it. In general it can be said that in acute depletion, such as may occur after massive hemorrhage, the chief consideration is the early replacement of the lost plasma protein. This can be accomplished best by repeated, early blood transfusion. Protein feeding, because of the nature of the underlying disease, frequently has to be postponed until well into convalescence. The administration of minimal amounts of fluid and sodium chloride is essential.

For the protein deficiencies due to simple starvation, the providing of adequate amounts of protein in the diet is usually sufficient. Animal protein is the food of choice.

In those instances where an insufficient protein intake is due to an underlying obstructing disease, such as ulcer or carcinoma, successful treatment depends upon a direct attack upon the fundamental cause of the condition, usually by surgical procedures. In addition, preoperative and postoperative care of the patient by repeated transfusions, limitation of fluid and salt and occasionally emergency feeding by jejunostomy constitute the necessary measures for complete success. Recognition of the problem of protein deficiency in relation to the "poor risk" patient will go far to increase the success of surgical measures indicated in such cases.

If improper absorption is due to mucosal changes, secondary to a condition such as beriberi, the treatment of the primary deficiency usually results in the successful management of the symptoms due to protein lack.

When there is protein deficiency due to a chronic disease, such as nephritis, cirrhosis, and so forth, therapeutic measures must consist largely in the administration of slightly better than maintenance amounts of protein in the diet and limitation of salt and fluids. Diuretics may at times be of temporary assistance in controlling the existing edema, and of these the mercurial preparations are the best. They are chiefly useful in cases of cirrhosis with low plasma protein and recurrent ascites. Transfusions are of only temporary help, but at times are indicated, even in the chronic cases of protein deficiency, in order to raise quickly the plasma albumin.

In summary, then, we can say that protein deficiency, acute or chronic, has as its causes: (1) an insufficient intake of protein; (2) failure of absorption; (3) an increased loss of protein from the body or, possibly, increased destruction or failure of regeneration of protein. Treatment consists, obviously, in providing an adequate protein intake whenever possible; in the specific treatment of any other deficiencies which may, at the same time, interfere with the proper absorption of protein; in appropriate surgery with proper precautions in instances where inadequate intake or insufficient absorption is due to a lesion of the gastrointestinal tract; or in the more chronic cases in the use of transfusions and diuretics in order, at least temporarily, to alter plasma protein values. In all instances it is expedient to limit fluid and sodium chloride intake.

CHAIRMAN MINOT: Deficiency of protein plays but a small role in producing a deficit in the body of the compound protein, hemoglobin. The clinical study of deficiency of this substance resolves itself largely into a study of iron deficiency. It has been known that iron was a specific for the "green sickness", chlorosis, since before the days of Sydenham and that diet could affect the amount of iron in the blood since Menghini in 1746 showed that it could be increased by feeding iron-containing foods. We are indebted to George H. Whipple for placing on a secure quantitative basis the influence of food on anemia. There remain, however, many mysteries about the metabolism of hemoglobin and iron. Certain facts have become clear, and among them is the actuality that iron deficiency is a dominant factor in the production of hypochromic anemia which appears clinically under various names, such as anemia of chronic blood loss, idiopathic hypochromic anemia and simple anemia of pregnancy. The last was included in Walter Channing's (our first Professor of Obstetrics) classic paper in 1842. These anemias that respond entirely satisfactorily to the oral administration of iron were included among the so-called secondary anemias in a less exacting medical era. Heath, with Strauss and Castle, was the first to demonstrate in hypochromic anemia that iron given by injection will be returned quantitatively as newly formed hemoglobin (10 milligrams of iron forming 3 grams of hemoglobin). Thus there is clear-cut evidence of direct participation of the deficient substance in the recovery following its administration. "The Mechanism of Hemoglobin Deficiency" will be presented by Dr. Clark W. Heath.

MECHANISM OF HEMOGLOBIN DEFICIENCY*

BY CLARK W. HEATH, M.D.†

FOR the purpose of this discussion I wish to apply the term "hemoglobin deficiency" to that type of anemia in which the formation of hemoglobin is particularly at fault, namely, hypochromic anemia.

Hemoglobin may be regarded as a protein in which a large molecule, globin, is linked with a smaller, iron-containing molecule, hematin. Under ordinary nutritional circumstances the body apparently is capable of furnishing on demand all the substances which go to make up

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the protein molecule, hemoglobin, with the frequent exception of iron. Even the pyrroles, which form an essential part of hematin, can apparently be manufactured in the body. But iron is, of course, an element that is unchanged and unchangeable and must be provided to the body from outside. Although it is extremely important to the body, it is not present in very generous amounts and is very easily exhausted, particularly when hemoglobin is lost by hemorrhage. When iron is given intramuscularly to cases of profound hypochromic anemia it can be recovered quantitatively in newly formed hemoglobin. This illustrates the peculiarly important place that iron has in the formation of hemoglobin. In the absence of a supply of iron, that is, in iron deficiency, hemoglobin cannot be manufactured.

Nevertheless, there are a number of substances that, in the presence of iron, have been shown to have an additive effect upon hemoglobin production. In Whipple's work will be found certain foodstuffs arranged according to their power of regenerating hemoglobin in dogs rendered chronically anemic by repeated bleeding and maintained on a diet poor in hemoglobin-regenerating factors. A particular liver extract, prepared by precipitating the aqueous extract with 70 per cent alcohol, has been shown to produce hemoglobin regeneration in these dogs. Bile pigment, chlorophyll and chlorophyll derivatives have been shown to be effective, when added to small doses of iron, in increasing blood regeneration in hypochromic anemia of man. Copper is apparently a necessary substance for hemoglobin formation, and this has been definitely proved in small animals. Although iron will be absorbed when given to anemic rats in the absence of copper, it will not be utilized to form hemoglobin until copper is added. Other metals, such as cobalt, nickel and manganese, perhaps have in very minute amounts, a similar influence to copper. The practical value of these substances in human hypochromic anemia is very doubtful. These substances, while aiding blood regeneration, do not seem necessarily to be supplying specific deficient factors of the hemoglobin molecule itself, but to be helping intermediate metabolic processes in the formation of hemoglobin. For example, the administration of suboptimal doses of iron in acid-buffered mixtures will cause greater blood production than in alkaline mixtures, probably by facilitating iron absorption in the small bowel.

In addition to the administration of these various substances that aid hemoglobin regeneration, there are innumerable factors that have important modifying effects. A certain healthy state of the body is necessary for the formation of hemoglobin and the maintenance of the normal hemoglobin concentration. Hemoglobin manufacture is a chemical process taking place continuously and is closely associated with other metabolic processes of the body. Thus, the absence of the thyroid secretion, deprivation of vitamin C and any diseased condition, such as chronic infection, nephritis or carcinomatosis, that interferes with metabolic processes of the body, including cellular function and formation in general, will interfere with hemoglobin formation. These various states frequently result in anemia, which may be hypochromic in nature and which signifies, therefore, an inhibition of the process of hemoglobin formation, though not on the basis of a deficiency of the essential components of hemoglobin. Unless these factors are adjusted properly, the administration of iron in cases of iron deficiency will fail.

A deficiency of iron in the body is common and, as noted, leads to hypochromic anemia. The anemia is, as a rule, quickly alleviated by giving inorganic salts of iron by mouth. Iron so given does not act as a so-called "stimulant", but supplies a deficiency, for iron is ineffective in other types of anemia not associated with iron deficiency. How the deficiency of iron comes about is not clear in every case, but certain general rules can be formulated. The body is very sparing of iron. There is no definite evidence that a negative iron balance, that is a loss of iron from the body, can be produced by limiting the amount of iron in the diet. Although Sherman has stated that 15 milligrams per day is an optimum iron intake, diets containing much less iron will maintain the iron balance. This is true even though only a fraction of the iron in the food is available to the body. The amount of iron excreted in the urine is practically negligible. Therefore, there is a marked difference between the metabolism of iron and the metabolism, for example, of such elements as nitrogen or calcium, for a loss of nitrogen or calcium may very easily be produced by restricting these substances in the diet. One must search for a loss or an increased consumption of iron to explain in large part an iron deficiency.

There are a number of mechanisms active during different periods of life by which iron may be lost or exhausted. In growth there is a demand for iron to supply in particular the increasing mass of hemoglobin in the expanding blood volume and also to supply the needs for iron of the tissues of the body. In pregnancy there is a demand for iron by the fetus. In women there is a continual demand for iron due to the blood loss accompanying menstruation. These factors of iron consumption and loss must play a primary role in the causation of hypochromic anemia, dietary deficiency of iron or malabsorption of iron in a diseased gastrointestinal tract being of secondary impor-

tance. This state of affairs is theoretically true in all dietary deficiency states. First in importance is the loss or increased consumption of the deficient substance and, secondly, inadequate intake.

The recognized types of iron deficiency anemia, some of which in the past have been regarded as of very obscure etiology, are as follows: hypochromic or nutritional anemia of infancy and childhood, chlorosis, "idiopathic" hypochromic anemia, hypochromic anemia of pregnancy and hypochromic anemia of blood loss.

Iron deficiency occurs in childhood probably because of the excessive demand of growth, inadequate iron endowment from the mother, diets poor in iron (for example, an exclusive milk diet during the first year of life) and gastrointestinal disturbances that interfere with absorption. It occurs in girls after puberty usually because of the combined demands for iron of growth and menstruation, together with diets poor in iron. The theoretical demand for iron in a year's time in a girl after puberty is approximately the same as the demand for iron of a normal pregnancy. Chlorosis, although much less common than in the preceding century, has not disappeared and, at least in mild form, is undoubtedly very common. Dr. Arthur J. Patek and I have observed in the past two years at least six cases of severe anemia in girls which seemed very typical of what could be called old-fashioned chlorosis. In "idiopathic" hypochromic anemia, which usually occurs in middle-aged women, careful clinical study of cases has shown almost without exception blood loss, usually by menorrhagia or bleeding hemorrhoids occurring in a subtle and chronic form, but some cases have had multiple and frequent pregnancies, diets poor in iron or gastrointestinal disturbances, associated with achlorhydria, that interfere with absorption of iron. The hypochromic anemia of pregnancy results from the demands of the fetus for iron, poor iron reserves, a diet poor in iron and malabsorption of iron. The demand for iron when there is blood loss is obvious. The effect of factors altering the internal metabolism of iron on the production of anemia requires study and is but one of many aspects of hemoglobin metabolism that remain to be solved.

It seems desirable and important to group all of the various types of hypochromic anemia under the term *iron deficiency*. There is no question that these forms of anemia represent a deficiency disease. Moreover, this is a deficiency disease in which the extent of the deficiency can be determined quantitatively at any time by the determination of the hemoglobin in the blood and in which the deficient factor, iron, can be supplied quantitatively. The various ways in which the deficiency arises are now fairly well understood. We have, therefore, data concerning this deficiency disease which we have not as yet been able to obtain concerning those deficiency states in which the deficient factors are organic substances and the manner of activity is little understood. If the clinician will bear in mind the fundamental causes of iron deficiency as seen in man, will eliminate, if possible, causes of blood loss and will treat such cases by the administration of proper doses of iron, there will be a very large number of people, especially women, with much better health.

Dr. Heath illustrated his communication by several lantern slides.

CHAIRMAN MINOT: Modern knowledge of nutrition has in large part developed by means of studies on normal animals in which dietary deficiency disease is produced by the elimination of specific substances in the diet. That deficiency disease in man may, and frequently does, develop because of some disturbance of the gastrointestinal tract in spite of an apparently adequate diet is of recent recognition. Dietary deficiency itself may lead to defects of the stomach and intestines, and thus a vicious cycle can arise.

A totally new concept for the origin of a deficiency disease was demonstrated in 1928 when it was shown that Addisonian pernicious anemia arose because of an abnormality of gastric secretion. Since then the knowledge of the relationship of the gastrointestinal tract to dietary deficiency disorders has advanced rapidly. The man responsible for the ingenious hypothesis and beautiful demonstration of the role that a lack of a gastric factor on the one hand and a dietary factor on the other plays in the production of pernicious and related macrocytic anemias has justly received recognition for his work from different parts of the world and in various ways. Dr. Castle is to be congratulated on his epoch-making contribution to clinical medicine and human physiology. He will speak to us on "The Relationship of Defective Nutrition to Changes in the Gastrointestinal Tract", regarding which he constantly adds information.

THE RELATIONSHIP OF DEFECTIVE NUTRITION TO CHANGES IN THE GASTROINTESTINAL TRACT*

(ABSTRACT)

BY WILLIAM B. CASTLE, M.D.†

AN important though obvious feature of nutrition is the fact that food, until it has left the alimentary tract, is still in a physiologic sense outside the body. The digestive tube is essential to the reduction of the raw materials of the food to the proper size and constitution for absorption. Failure of this normal function of the alimentary tract may thus "condition" a deficiency of water, salts or energy-producing material. Certain vitamin deficiency diseases are also apparently produced by gastrointestinal defects. Indeed, the function of the alimentary tract may be specifically concerned in the metabolism of an accessory factor in the food. For example, during the past decade, studies of the macrocytic anemias that respond to suitable liver extracts have disclosed a particular function of the normal stomach in respect to blood formation. Thus, a factor in the food normally becomes effective in the body as "liver extract" only after contact with a substance secreted by the stomach. Disturbances of the function of the alimentary tract, even of certain portions of it, may thus have a general or a subtly specific effect upon the nutrition of the individual. Once established, certain disturbances of the digestive tract may "condition" the individual for a dietary deficiency disorder even though he partakes of a diet completely adequate for normal man.

Digestive disturbances since the dawn of medical knowledge have been known to result from improper food. About fifteen years ago McCarrison in India showed that widespread changes of the alimentary tract in animals can be produced by defective nutrition. It is now becoming recognized that defective diets may produce in man degenerative defects, at least of certain portions of the alimentary tract. Conditions associated with changes, temporary or permanent, in the tongue, stomach and intestines are apparently due in many instances to chronic dietary deficiency. They are clinically manifest in pernicious anemia, sprue, pellagra and in less well-defined conditions frequently seen in hospital and private practice. Thus an adequate diet is essential lest irreparable injury be done to the organ that conditions the raw material of the food before it is converted into the nutrient medium of the body.

CHAIRMAN MINOT: The history of scurvy forms a fascinating chapter in the history of the world and science. One should not neglect to enjoy the description by Jacques Cartier in 1536 of the cure of scurvy "by drinking in six days a (spruce) tree *twice* as large as a French oak", nor should one fail to read the records of the *controlled* experiments in 1747 by James Lind showing that orange or lemon juice is a specific cure. It was not until about 1919 that scurvy was generally accepted as a deficiency disease due to the lack of a specific chemical substance. Only recently has vitamin C (cevitamic acid) been isolated and synthesized. The importance of characterizing the vitamin deficiencies pathologically was appreciated by Dr. Wolbach more than twelve years ago. He was the first to point out that repair phenomena during recovery from vitamin deficiency must be studied. Recently he has noted that we have in the study of vitamin deficiencies an opportunity, for the first time in the history of biology, to correlate intracellular chemistry and cellular morphology. Dr. Wolbach with Dr. Howe has made numerous important contributions to the pathology of vitamin deficiencies. Their most significant discovery is the characterization of scurvy as a disease in which the whole pathologic anatomy can be based on the consequences of the deficiency of intercellular material. "Vitamin C and the Formation of Intercellular Material" will now be presented by the Shattuck Professor of Pathological Anatomy; Dr. Wolbach.

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VITAMIN C AND THE FORMATION OF INTERCELLULAR MATERIAL

BY S. BURT WOLBACH, M.D.*

THE name vitamin connotes a preformed substance necessary for some essential intracellular chemistry in the body. Because no one today has defined vitamins, it is pertinent to state that the substances so designated are all directly or indirectly of plant origin. They are not sources of energy and are not building materials as are fats, carbohydrates and proteins. Deprivation of a vitamin suppresses a physiologic process. The cells concerned may exhibit structural changes, but may survive and be capable of growth and multiplication. Such effects are easily demonstrable in epithelial cells in vitamin A deficiency, in cartilage cells in vitamin D deficiency and in all supporting tissue cells in vitamin C deficiency.

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Vitamin C deficiency, of all those studied, produces the most striking alteration in the body structures. Because its chemical structure and some possibilities of its function within cells are known, I take this occasion to present again the consequences of this deficiency, which affords the best promise of a complete solution of any of the vitamin deficiency phenomena—a solution of great importance for progress in the physiology and pathology of intercellular materials, notably the so-called degenerative diseases of blood vessels and joints and even the processes characteristic of old age.

Vitamin C (ascorbic or cevitamic acid) is necessary for the formation of all intercellular substances having collagen as their basis, and its absence prevents the formation of the matrices of white fibrous tissue, bone, cartilage and dentine. This explains all gross lesions of scurvy. The consequences of its absence are therefore most apparent in growing animals and in the repair of lesions of the above tissues. The deficiency does not prevent multiplication of the cells of each type of tissue, but the cells assume a common morphology, approximately that of embryonic connective tissue, and histologic appearances indicate that a liquid material is produced in lieu of the normal matrix. Administration of ascorbic acid by mouth or parenterally is promptly followed by the production of intercellular substances and by the resumption of cytology characteristic of the tissue concerned. This method of suspending and inducing collagen formation in growing animals and in experimentally created defects permits study of the sequence of formation and differentiation of intercellular materials and has definitely proved that collagen is a secretory product of the cell, in exclusion of several theories of its origin.

Extended studies of guinea pigs in absolute vitamin C deficiency and of others maintained for long periods on inadequate ascorbic acid rations have failed to reveal morphologic evidence of loss of function of epithelial tissues, but have given evidence of a quantitative relationship between the amounts of vitamin C administered and the quantity of intercellular materials produced. Biochemists, in attempting to apply the properties of ascorbic acid to physiology essential for the organism, have failed to realize that there is at least one recoverable end product dependent upon its presence. Because cell division occurs in absolute vitamin C deficiency and, therefore, the continuation of synthesis of proteins, the speculations of a morphologist, irresistibly, though timidly, are along the lines of attributing to ascorbic acid a late role in the formation of collagen, possibly the polymerization of a simple molecule hazily in mind as something like gelatin or whatever the apparently liquid substance is that the mesenchymal cells produce in scurvy.

Dr. Wolbach then demonstrated a number of lantern slides of tissue sections illustrating the pathologic effects of vitamin C deprivation.

CHAIRMAN MINOT: In my introductory remarks I alluded to the importance of detecting and correcting suboptimal nutrition. Deficiencies are relatively easily produced during growth, so one may expect to observe the effects of partial dietary deficiency more often in children than in adults. Disorders from improper food may appear to be slight, yet under such circumstances they may slowly lead to altered function and anatomic changes that permit organisms or other factors to be the more immediate cause of disease. There is no phase of medicine more essential than preventive pediatrics. The early correction of any nutritional defect in the child is of the utmost importance. Slightly improper nutrition during growth may cause defects many years later and a shortening of the span of life. The advances being made in different fields of medicine and biology are aiding to detect incipient deficiency states. Dr. Blackfan, with Dr. Wolbach, has contributed information concerning the early diagnosis of vitamin A deficiency. Histologic changes may develop in the tissues before external symptoms are obvious. Studies concerning the early recognition of vitamin deficiency are being actively pursued in various departments of the University. The Thomas Morgan Rotch Professor of Pediatrics, Dr. Blackfan, will now tell us of the progress of knowledge in this important field.

PROGRESS IN THE EARLY RECOGNITION OF VITAMIN DEFICIENCY STATES*

BY KENNETH D. BLACKFAN, M.D.†

IN this consideration of the progress in the early recognition of vitamin deficiency states, it would seem appropriate to recall the many milestones which have been passed since a number of clinicians,[1, 2, 3, 4, 5] whose writings have become classic, clearly described the advanced clinical manifestations of rickets, scurvy, beriberi, xerophthalmia and pellagra. Attention was directed by these writers of medical history to the chief diagnostic features in each one of these diseases—namely, in rickets, to beading of the ribs and deformities of the limbs; in scurvy, to bleeding of the gums and painful extremities; in beriberi, to a paresis of the limbs and a tachycardia with terminal cardiac failure; in xerophthalmia, as the name implies, to a peculiar dryness of the conjunctivae which led to blindness and which was associated with malnu-

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trition and stoppage of growth; and in pellagra, to a glove-like dermatitis of the hands, a red tongue and gastrointestinal disturbances. Therefore the late clinical manifestations of these disorders have been known for a long time.

Notwithstanding these contributions, the theories as to the etiology of these clinical entities were for many years wholly speculative. It was intoxication. It was infection. Many other fanciful suggestions were entertained. When it was finally agreed that the true nature of these disorders rested on a dietary basis, it became possible to prevent and cure them empirically. Nevertheless, the actual errors in the diet remained a mystery until new light on this subject was shed through experiments on small laboratory animals.

It has been said that Lunin[6] in 1881 first demonstrated that white mice would not thrive on a purified diet containing the "proximate principles" in milk. He thought the diet was so unpalatable and monotonous that the mice lost their appetite and inevitably died of malnutrition. The significance of this observation was not fully appreciated until Sir Frederick Gowland Hopkins[7] confirmed these experiments His singular contribution was the demonstration that full health was restored when a *small amount of milk* was added to the purified diet. Shortly it was found that the effectiveness of adding fresh milk to a purified diet was due to a number of "hitherto undescribed" essential dietary factors. The term, *vitamin*, used popularly to indicate certain of these food substances, which even in minute amounts make the difference between health and disease, was introduced in 1912.[8] Thus the "deficiency" era was begun.

It has become known in this way that in a diet adequate for the preservation of health and the prevention of disease there is needed a correct proportion and a sufficient amount of water and the chemical component parts of protein, fat, carbohydrate, minerals and vitamins. The necessary nutritive substances must not only be contained in the food, but they must be present in a digestible form and their absorption and utilization must not be interfered with by underlying pathologic processes.

We have much to learn about the limits within which the body can adjust itself to variations in intake of the different vitamins, but within these limits not every deficiency brings about discernible alteration in function. It is only when the limits of safety are grossly overstepped that the morphologic and physiologic effects become detectable by clinical means.[9]

It is not surprising to find that following each new experimental discovery in this field, physicians have been stimulated to search by the use of refined diagnostic measures, applicable to man, for early evidences indicating a vitamin deficiency. In vitamin A deficiency, there has developed the photometer test,[10] and keratinized epithelial cells in the urine and in scrapings from the cornea are found before the diagnostic sign, xerophthalmia, makes its appearance.[11, 12, 13] In vitamin B deficiency, no very early test has proved acceptable before the pathognomonic signs suggestive of beriberi or pellagra become noticeable. For vitamin C deficiency, it is clear that the x-ray may reveal changes in the long bones before the beaded ribs and the bleeding gums are observed.. So too for vitamin D deficiency, the x-ray may portray the characteristic cupping and fraying of the long bones before the rosary or bowed legs are apparent.

Today, as the consequence of the recent advances concerning the chemical and morphologic nature of vitamins, it may be said that we are entering a new era—an era of recognizing *still earlier* states of vitamin deficiency. Quantitative chemical tests are at hand ready for clinical application, whereby the concentration of vitamins A, B_1, C and D can be measured with a certain degree of confidence. By them it should be possible to appraise not only the nutritional status of the individual with respect to meeting the optimum vitamin requirements, but also to detect the presence of more than one vitamin deficiency in the same individual.

Time will not permit a discussion of the chemical and morphologic characteristics of the vitamins, so we will pass directly to the more salient features of the quantitative chemical tests for vitamins A, B_1, C and D.

For vitamin A, the antimony trichloride test is the one best suited for clinical purposes. Carr and Price[14] found that antimony trichloride produced a blue color with vitamin A. The average value in man on an adequate diet as determined by the Lovibond tintometer is 40 blue units per 100 cc. of serum or approximately 0.05 milligrams per cent of vitamin A. It has been found that the average normal concentration of vitamin A varies with the diet and with advancing age.[15] The unitage is very low in severe infections. With proper precautions and careful selective measurement of color intensity, this is a simple, rapid quantitative test for the analysis of vitamin A in blood serum and tissues.

Turning now to vitamin B_1, several chemical tests have been suggested which, it is believed, will serve a useful purpose. These are the estimation of bisulfite binding substance, methyl glyoxal and pyruvic acid.[16, 17, 18] Each of these requires further study to determine its clinical applicability. Concerning the pyruvic acid test, Peters[19] has shown that in vitamin B_1 deficiency there is a defect in oxidizing certain carbohydrate intermediates in nervous tissue—in particular, pyruvic acid. Vitamin B_1 is the factor

that serves to promote its oxidative removal. Pyruvates have been found in the blood of patients with beriberi in China, and the blood of experimentally depleted animals contains pyruvic acid in relatively large amounts. The feeding of vitamin B_1 in the diet of depleted animals causes pyruvic acid to disappear and brings about restoration to full health.

The chemical test for vitamin C is the titration method with the dye, indophenol blue, which forms a colorless compound with ascorbic acid. This test is widely used for the analysis of ascorbic acid in urine and tissue. The determination of ascorbic acid in the blood awaits further confirmation before its actual significance can be stated. With a barely adequate diet, 20 to 40 milligrams of ascorbic acid are excreted in the urine daily. A series of orally administered test doses of 200 milligrams of ascorbic acid provides valuable information as to the state of vitamin C nutrition, as surplus ascorbic acid is not excreted until adequate storage in the tissues is present. After "saturation" has been reached, 75 per cent of a 200 milligram dose is excreted in the urine within eight hours of ingestion. The average normal value in the blood is 0.8 to 2 milligrams per cent and in the liver is 15 to 30 milligrams per cent.[20, 21, 22, 23]

Figure 1 illustrates the ascorbic acid content in the urine and blood from an infant with advanced scurvy. Note the ascorbic acid values for urine in the left column. The values for blood are indicated in the next column. On admission, there was a negligible quantity of ascorbic acid in the urine and blood. Five doses of 200 milligrams of the crystalline vitamin were administered by mouth before urinary excretion was perceptibly affected. Another 1000 milligrams were given before urinary values were increased to 40 per cent of the amount ingested and before the ascorbic acid values in the blood approached the normal level.

It is interesting to point out the close correlation between the chemical tests for vitamin C deficiency and its histologic counterpart. In studies of the liver tissue in twenty-five unselected infants at autopsy, Ingalls[24] found that the ascorbic acid value in six was distinctly below the estimated normal values for liver tissue—15 to 30 milligrams per cent. In these six patients the ascorbic acid values varied between 2.7 and 5.3 milligrams, and in each of these patients studies revealed the characteristic histologic changes of early scurvy. In another patient who presented both clinical and roentgenologic evidences of acute scurvy during life and the histologic changes of scurvy at necropsy, the ascorbic acid content of the liver was negligible.

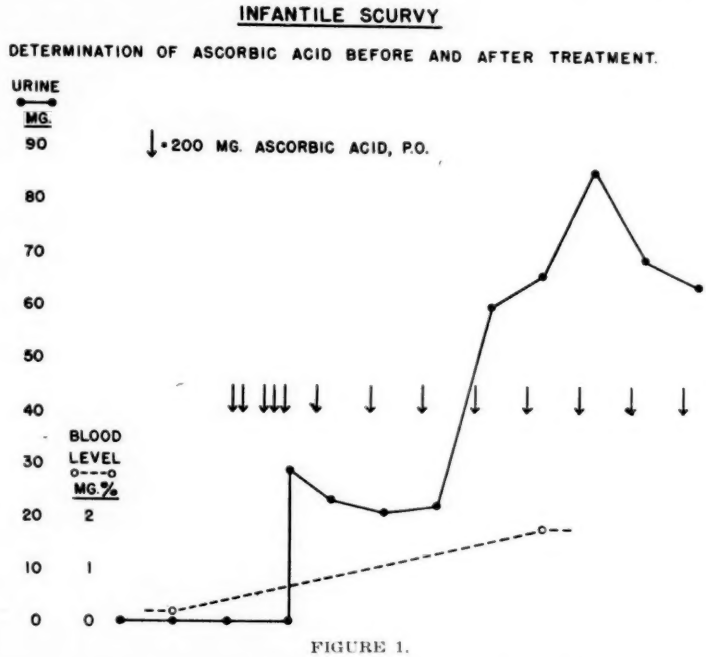

FIGURE 1.

Before rickets is recognizable clinically, the determination of calcium and phosphorus in the blood serum indicates that optimal amounts of vitamin D are not available. A serum phosphorus of less than 3 milligrams per cent and a calcium and phosphorus product of less than 40 indicate moderate or severe rickets. It should be emphasized that the calcium and phosphorus figures require evaluation as to activity of growth and with the level of serum protein.

It has been claimed that the most sensitive test for detecting active or current rickets is found by measuring the phosphatase in the blood. In the beginning of the disease it is increased and does not return to normal until healing is

complete. In this respect it is a more reliable and sensitive index than the information secured from the calcium and phosphorus product and the x-ray. Further information is needed to determine the significance of phosphatase as an indication of early vitamin D deficiency.[25, 26]

MULTIPLE VITAMIN DEFICIENCY

RICKETS AND SCURVY

Patient: A 4 mos. old female. Wt. 11 lbs.

Diet: Cow's milk formula.
Never given cod liver oil.
Never given orange juice.

Physical Signs: Anemia; tenderness of limbs.
Beading of ribs; craniotabes.

Roentgenogram: "Somewhat suggestive of scurvy."

	Chemical Tests	
	Admission	*Discharge*
Ascorbic Acid (Blood)	0.0 mg. %	0.45 mg. %
Calcium	8.2	10.4
Phosphorus	3.4 = 27.88 P	4.6 = 47.84 P
Phosphatase	0.29 Kay units	—

FIGURE 2.

In figure 2, data are given illustrative of a multiple deficiency state. An infant of four months was studied with equivocal symptoms and physical evidences and roentgenologic signs of rickets and scurvy. Note the chemical tests, as they suggest the presence of both rickets and scurvy. On admission the ascorbic acid in the blood was 0, the calcium and phosphorus product was 27.88 and the phosphatase test was high normal—0.29 units. Treatment for rickets and scurvy was followed by a return of ascorbic acid to 0.45 milligrams per cent with a calcium and phosphorus product of 47.84.

To reiterate, we have ready for clinical application chemical tests for the detection, quantitatively, of four specific vitamins. For vitamin A there is the antimony trichloride test; for vitamin B_1 further work is needed to determine the value of the pyruvic acid test; for vitamin C there is the indophenol test; and for vitamin D there is the phosphatase test, as well as the calcium and phosphorus measurements of the blood. In conclusion it should be emphasized that much remains to be done. As in the past, every new technical procedure has required modifications or adaptation for ease of performance and precision before it has become practical and applicable for the study of disease in man. So with the chemical tests for vitamins it will be necessary to accumulate a vast number of data for setting up accurate standards in health and in disease to serve as guideposts by which states of vitamin deficiencies may be detected earlier than has been possible heretofore.

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CHAIRMAN MINOT: A new day has dawned for dentistry—a day of organized research, and one of great importance to human welfare because the prevention of dental disorders is the prime object. Dental problems should be studied in relation to man as a whole, and thus concern all branches of medicine. There is no better example of this than the effect of diet on teeth and their adjacent structures. Dr. Howe, in 1914, was the first person in America to carry out experimental observations concerning this subject. He has indicated that subclinical scurvy can be responsible not only for defective dentition but also for most types of malformation of the bony structures of the mouth. In 1924, Dr. Robert W. Lovett, as a member of the Board of Trustees of the Forsyth Dental Infirmary, appreciated that Dr. Howe's

interests would be advanced by a pathologist, which led to Dr. Wolbach's and Dr. Howe's successful endeavors to characterize vitamin deficiencies pathologically.

Dental caries is rare in primitive people and is to be associated with so-called "civilization". The incidence of caries in children may be reduced by regulation of the diet as Dr. Howe, among others, has demonstrated. Oliver Wendell Holmes would certainly be pleased to realize that as one of the real founders of dental education at Harvard he had helped pave the way for the recognition that the development of dentistry and medicine must go hand in hand and be increasingly closely associated. "The Relation of Avitaminosis to Oral Pathology" is a significant topic. The Thomas Alexander Forsyth Professor of Dental Science, Dr. Howe, will speak to us on that subject.

THE RELATION OF AVITAMINOSIS TO ORAL PATHOLOGY

BY PERCY R. HOWE, D.D.S.*

THE most common pathologic lesion of the oral cavity is decay of the teeth. Until 20 years ago dental caries was supposed to be due to the fermentation of carbohydrate material with the formation of lactic acid. The acid was supposed to eat its way into the tooth, forming the cavity. Bacterial examination of carious material showed lacto-bacilli to be present in the cavity. However, no one has as yet succeeded in producing caries in animals by means of these bacteria or by feeding fermentable sugars or starches. Added to this is the widespread prevalence of dental caries, the incidence of which is estimated by competent authorities to be about 95 per cent among school children. Doubt has arisen as to the entire validity of this theory. Caries has, however, occurred among animals on diets deficient in vitamins and in calcium or phosphorus. The precise cause remains obscure and affords a subject for further study.

Pyorrhea is nearly as common as caries, occurring more often in adults than in children. The bacterial aspect of this disorder has also been relegated to a secondary position. It is generally conceded that pyorrhea is not a distinct disease entity but is a symptom of an anomalous metabolic condition. Lesions similar to it follow in scorbutic or rachitic feeding.

The incidence of both caries and pyorrhea is as great, if not greater, than it was 50 years ago.

Disturbance of the normal growth and development of the bones that form the structure of the oral cavity, resulting in irregularity in the position of the teeth and of the constriction of the nasal passages, has followed in animals when they have been deprived of vitamin C or D.

The effects of a deficiency of vitamin A, C or D on tooth structure during the formative period have been described in detail in the literature. It is well understood that the most striking effects are produced early in life and that they may be carried so far as to result in permanent deformity of calcified tissues.

The temporary teeth begin to calcify at about the third fetal month. Caps of the permanent teeth are found at five and one-half months. The mandible, from a single center of ossification, is the first bone to calcify with the exception of the clavicle. The maxillae begin to ossify from six centers of calcification at from the sixth to the eighth fetal month. The process continues rapidly during infancy and young childhood. Interruption of the normal process is followed by defective teeth and irregularity in their arrangement. At this time in life these matters are in the hands of the medical practitioner. The dentist sees the teeth only after their eruption. His efforts are mainly reparative or corrective. He is fitted neither by training nor by experience to give authoritative advice on nutritional matters.

While there is much that is obscure concerning dental conditions and, while the need for protracted investigation is self-evident, sufficient evidence has been disclosed to indicate that the dental disaster of today is largely due to mineral and vitamin deficiencies.

There is some evidence to be seen in vitamin studies that supports the finding of diffraction studies, in that unsuspected changes occur in the organic substance of teeth and bones in such a condition as rickets. The organic substances of the teeth have not been studied to any extent and await investigation. The whole subject of tooth structure was disregarded until the studies of Mrs. Mellanby, Zilva, Wells and some investigators here began to show that tooth structure and dental caries were associated.

Vitamin investigation has clearly shown that the teeth do not stand apart from the rest of the body but that, on the contrary, they are intimately associated with its physiological processes. The same pathologic response to vitamins A, C, or D deficiency and to recovery therefrom on furnishing the deficiently fed animal with the missing factor is seen in the teeth as elsewhere.

While absolute deficiency of these vitamins enables the student to study the cellular performance in tissue in a clear-cut fashion, in clinical practice latent, protracted and often unrecognized effects are encountered. These are at the moment under investigation, and it is prob-

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able that the dental manifestation of such states is what is so often observed.

The few facts that have been disclosed regarding the effect of food deficiencies on the teeth have already been put to clinical tests with notable dental improvement, as well as improvement in the general physical condition.

The effect of vitamins on other oral tissues cannot here be described because of the limited time at my disposal. Nor can it be undertaken to indicate special subjects for future research, except to state that for a fuller understanding of the modus operandi of vitamin activity in its relation to oral pathology the work must of necessity lead off into what may seem to be far afield.

It is the hope of present day research workers that medical men will work to build good dental conditions by advising proper nutrition in prenatal and early postnatal periods, in so far as this is understood, and that dentists will regard dental pathology not as an isolated situation, but as the result of general physiologic conditions. Only with the cooperation of the medical profession can dentists hope to eliminate such oral pathology as is apparently due to vitamin and mineral deficiency.

Dr. Howe then demonstrated a series of lantern slides showing, especially, bone defects from dietary deficiency.

CHAIRMAN MINOT: Defective nutrition can be responsible for nervous and mental disorders. Such conditions can arise from deficiency in the caloric intake as well as from deficiency of certain specific diet factors. The serious nerve lesions of beriberi, pellagra and pernicious anemia are to be attributed to lack of food-derived substances. Animal experimentation has shown the disastrous effects of avitaminosis on the nervous system.

James Jackson, our second Hersian Professor of the Theory and Practice of Physic, in his classic description of alcoholic polyneuritis noted that these patients suffer from disordered digestion and that in treatment "animal food was most useful". Somewhat over 100 years later Harvard investigators, recognizing the similarity of the symptomatology and pathology of beriberi and of alcoholic polyneuritis, gave evidence that dietary deficiency plays an important role in the production of polyneuritis in chronic alcoholic cases. That the polyneuritis of pregnancy is a dietary deficiency disorder was first shown definitely by Dr. Strauss and Dr. William J. McDonald. At Harvard it was also first shown that the neural lesions of pernicious anemia could be entirely prevented by liver therapy. Dr. Strauss has been intimately associated with these various studies concerning "Nerve Disorders Arising from Defective Nutrition", about which he will speak to us.

NERVE DISORDERS ARISING FROM DEFECTIVE NUTRITION*

BY MAURICE B. STRAUSS, M.D.†

THE concept that nerve disorders might arise from defective nutrition may properly be said to have originated in 1897 with the work of the great Dutch investigator, Christiaan Eijkman. At that time, however, the brilliant bacteriologic researches of Pasteur, Koch and their followers so overshadowed other developments in medicine that physicians became imbued with the thesis that only positive agents—the bacterium, the parasite, the toxin—could cause disease. Hence Eijkman, although he had clearly established the fact that polyneuritic beriberi was due to a deficiency of something in food, was unwilling to advance so heretical a doctrine and, instead, suggested that foods such as polished rice, being overrich in starch, produced a substance in the intestine which was "poisonous" to nerve cells and for which the outer layers of rice acted as an antidote. During the past forty years, however, the further researches of Eijkman and those of Vordermann, Grijns, Hopkins, Strong and McCarrison, among a host of others, have firmly established the fact that polyneuritic beriberi is the result of a dietary deficiency and that the substance missing is a portion of the vitamin B complex.

I plan to discuss today three nerve disorders which, like beriberi, have long been considered of toxic origin—"alcoholic" polyneuritis, "toxic" polyneuritis of pregnancy and subacute combined degeneration of the spinal cord.

The use of alcohol appears to be of considerable antiquity. Certainly it was known to the son of Noah, if not earlier, and throughout the years there has been a belief that its use is sinful. It is therefore quite understandable why John Coakley Lettsom and James Jackson, over a century ago, upon first describing what is now commonly known as alcoholic polyneuritis, should have considered the nerve injury due to the alleged poisonous effects of alcohol. However, observations in recent years have shown that the clinical findings in this condition are in no way distinctive for users of alcohol. Even minute neurologic examination fails to differentiate "alcoholic" polyneuritis pregnancy polyneuritis, polyneuritis arising in cases where intestinal absorption is severely handicapped and beriberi polyneuritis, although it is a comparatively simple matter to distinguish the obviously "toxic" polyneuritides, such as those due to lead, tri-ortho cresyl phosphate and diphtheria toxin. Moreover, neither

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gross nor microscopic studies of peripheral nerves and spinal cords reveal differences between the first named polyneuritides, although it is not difficult to differentiate them pathologically from the toxic group.

If, then, alcoholic, pregnancy, and beriberi polyneuritides are clinically and pathologically identical, is there not perhaps etiologic identity? Several years ago Drs. Minot, Cobb and I first considered the food intake of patients suffering from "alcoholic" polyneuritis. The difficulties involved in obtaining adequate dietary histories from individuals who customarily pay more attention to, as well as more money for, ardent spirits than food need not be enlarged upon here. In 53 such patients, however, satisfactory data were obtained. In 2 of these patients the usual diet could be considered adequate, if not optimal, for nutrition. The remaining 51 patients had eaten for at least months, and frequently over a period of years, only very little fresh food of any kind and almost always sparsely of protein foods. In general it is this latter class of foods that supplies the bulk of the vitamin B complex in human diets. The caloric intake of these patients was supplied chiefly by concentrated carbohydrate foods and alcohol. The reason for the limited dietaries of these individuals was threefold. First, their economic circumstances were such that there was rarely enough money for both food and drink; food, to them, was of lesser importance. Secondly, the prolonged excessive use of alcohol frequently resulted in an irritative gastritis associated with anorexia. Thirdly, since a quart of whiskey contains at least 2800 calories, their total caloric intake was frequently high without the addition of other food.

From data on mice, rats, dogs and pigeons, Cowgill has determined that the vitamin B requirement is a function of both the weight of the subject and the total daily caloric intake. Although many of our patients may have taken sufficient vitamin B for their caloric intake of food, their requirement for this vitamin was greatly increased by virtue of the large number of calories which they imbibed as alcohol. Recently Jolliffe and his associates have corroborated our observations and have shown that although 18 of their 26 alcohol addicts with polyneuritis had an adequate vitamin B intake when the calories from alcohol were neglected, all were deficient when these calories were included in the calculation.

The second consideration in the study of patients with alcoholic polyneuritis was the state of the gastric secretions, since there is considerable evidence that virtual deficiencies of nutrition may arise in spite of an adequate diet if gastrointestinal defects are present. Gastric analyses were performed on 50 patients, of whom only 10 secreted normal amounts of free hydrochloric acid.

If the multiple neuritis of the alcohol addict is due to a nutritional deficiency, and particularly to a lack of some portion of the vitamin B complex, one would anticipate that a certain number of these patients would have manifestations of deficiencies of other nutrient factors, especially other parts of the B complex. It is hence of interest that 17 of our 67 cases had dermatitis and other lesions characteristic of pellagra, 12 had edema, and, in 1, signs of acute myocardial failure vanished rapidly with no other therapy than rest in bed, a little food and large amounts of a concentrate of the vitamin B complex.

These data, then, confirmed the hypothesis, first advanced by Shattuck ten years ago, that the multiple neuritis of the alcohol addict is a dietary deficiency similar to polyneuritic beriberi, and not the result of a direct toxic action of alcohol. Conclusive proof, however, could only be furnished by demonstrating that these patients would recover while they continued to consume their usual daily quota of alcohol, if they were supplied with an adequate intake of food factors. Accordingly, 10 patients with uncomplicated "alcoholic" polyneuritis were selected for study. By careful questioning the usual daily intake of spirituous liquor was determined in each case. Such an amount (from 1 pint to 1 quart) of whiskey was then administered daily to each patient throughout the period of study. At the same time each patient received, as a daily minimum of food, 2 eggs, 500 cc. of milk, 200 gm. of beef or lamb, 240 cc. of orange juice, 4 servings of green vegetables, 90 gm. of brewer's yeast or its equivalent and, by intramuscular injection daily, a vitamin B concentrate. Without exception the polyneuritis of each of these 10 patients was relieved over a period of 2 to 18 weeks during the constant administration of large quantities of whiskey. It is thus apparent that the ingestion of strong alcoholic beverages, up to a quart a day, has no demonstrable neurotoxic effect on peripheral nerves in patients receiving an adequate diet and vitamin B supplements. Such data, however, can be construed, just as Eijkman did his, to indicate that vitamin B or some other dietary factor serves as an antidote for the otherwise poisonous effects of alcohol. The data likewise do not eliminate the rather remote possibility that an impurity in the beverage alcohol consumed prior to the onset of the polyneuritis was responsible for the nerve lesion. However, they clearly indicate that the administration of large amounts of pure blended whiskey in no way prevents the relief of "alcoholic" polyneuritis when the patients are adequately nourished.

The "toxic" polyneuritis of pregnancy is a condition clinically and pathologically identical

to alcoholic and beriberi polyneuritides. Of the cases reported in the literature concerning which the data are available, all but a few have occurred in women who had had persistent, so-called "pernicious" vomiting over a period of weeks or months, as first noted by Whitfield. It is apparent that, irrespective of the intake, vomiting may result in severe dietary deficiency. Furthermore there is a general tendency to administer concentrated carbohydrate foods to such patients. In pregnancy it has been demonstrated that gastric secretory defects are the rule and may play a role in conditioning nutritional deficiencies. Furthermore, since it has been shown that the vitamin B requirement is directly proportional to the metabolic rate, the 20 per cent increase in metabolism that is of normal occurrence in pregnancy must increase the demand for vitamin B proportionately. Finally, and of greatest significance, is the fact that there are few recorded instances of recovery from the polyneuritis of pregnancy during gestation when treatment along conventional lines has been employed, whereas almost all the patients have recovered when they have received adequate supplements of vitamin B and other dietary factors.

Subacute combined degeneration of the spinal cord may perhaps occur independently but is usually associated with two known deficiency diseases, pernicious anemia and pellagra. Gildea, Castle and their associates, Mellanby and others have shown that animals fed on deficient diets may develop spastic paralysis associated with degeneration of the spinal cord, which bears certain similarities to the disease in man. Since subacute combined degeneration of the spinal cord associated with pernicious anemia may progress to a fatal termination, in spite of the fact that the patient is receiving sufficient therapy to maintain the blood at an approximately normal level, it has frequently been assumed that the cord lesion resulted from some unknown toxic factor and could not be influenced by therapy directed at supplying a nutritional lack. However, a series of studies carried on here during the past five years has demonstrated that subacute combined degeneration of the spinal cord treated by the intramuscular injection of liver extract in suitably large amounts can be entirely arrested. One cannot expect nerve cells and fibres in the central nervous system to regenerate once they have been completely destroyed, but in every instance the process of destruction has ceased and there has been no recurrence. The degree of improvement in the patient's condition that occurs once the degeneration has been arrested will depend on various factors, but not infrequently bedridden patients have been returned to their normal occupations. It is to be emphasized that the amount of liver extract employed must be sufficient for the individual case, no matter how much this may exceed the average dose. These data, then, point strongly to the hypothesis that the spinal cord lesion under consideration is the result of the deficiency of some factor that is present in liver extract.

In summary, then, it may be said that the researches of the past decade outlined above suggest that at least three nerve disorders, "alcoholic" polyneuritis, pregnancy polyneuritis and subacute combined degeneration of the spinal cord, each formerly considered of toxic origin, arise from defective nutrition, and may be successfully treated by supplying the deficient material.

CHAIRMAN MINOT: We have learned this afternoon particularly about nutritional disorders dependent on the lack of specific food substances. Nutritional defects can arise because of an inability to utilize food substances owing to a defect in some endocrine mechanism as takes place in diabetes. Dr. Joslin's classic book on this subject indicates the triumphal march he has watched and shared in since he graduated from this school 41 years ago—fully as active today as then. He has been alert, investigating and adding knowledge during the periods which he wisely designates as the Naunyn, the Allen and the Banting and Best eras of diabetes. An acknowledged master and leader in the field of diabetes, he once more proves himself responsive to what is new and serviceable. He was the first in this country to take up the study of Hagedorn's discovery of the benefits of a compound of protamine and insulin which was first publicly announced in Copenhagen about one and a half years ago. Dr. Elliott Proctor Joslin will tell us about the rapidly developing knowledge of "Protamine Insulin and Its Advantages."

PROTAMINE INSULIN AND ITS ADVANTAGES

BY ELLIOTT P. JOSLIN, M.D.*

THE most notable advance in the treatment of diabetes since the introduction of insulin in 1922 has been the introduction of protamine insulin by Hagedorn of Copenhagen. Wonderful as were the results achieved by the use of insulin, made available to the world by the Toronto investigators, Banting and Best in association with MacLeod and Collip, the handicap remained that the duration of its action was comparatively short and for many cases of diabetes three or four injections a day were required. To overcome this handicap various procedures were tried in the hope of delaying absorption, such as the insertion of the needle into thick adipose tissue, the mixing of insulin with gum arabic, various proteins, oil or lecithin, its combination with pituitrin or adrenalin, and finally with ferric chloride and tannic acid.

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None of these proved practical, but Hagedorn succeeded by precipitating insulin with a monoprotamine derived from the sperm of fish and then adjusting the pH of the mixture to conform to that of the body. In this ingenious fashion the absorption of active insulin from the subcutaneous depot was delayed and the length of action of insulin was doubled. This helped to open the way for still further improvements by which zinc or calcium were introduced into protamine insulin, which prolonged its action for over twenty-four hours and thereby made possible the treatment of nearly all diabetics requiring the use of insulin with injections but once a day.

Simplicity of treatment is now assured. The technic of diet and insulin is easily mastered. With the carbohydrate content of the diet about 150 grams, protein suitable to the age, weight and physical condition of the patient, and fat for maintenance of nutrition, it is easy to instruct a patient what to eat. Thus, with a slice of bread and butter and an orange or their equivalents at each meal, a moderate portion of cereal, 5 and 10 per cent vegetables in liberal quantities, and milk one-half pint and cream one-quarter pint or the reverse of the same, one has 150 grams of carbohydrate. As for insulin medication, one can start with protamine zinc insulin 10 units the first day and increase 10 units daily up to 40 units, unless before that time it is evident the urine is becoming sugar-free. If this is not the case, regular insulin can be added by separate injection at the same time as the protamine insulin before breakfast, beginning with 5 units and increasing the dose by 5 units daily. Seldom will it be necessary to raise the regular insulin above 20 units and protamine insulin 40 units in order to control the diabetes.

The secret of success in the use of protamine insulin is the recognition that it acts much more slowly than regular insulin. Its ultimate and good effect may not show for several days, a week or even longer. One must allow time to find out what its capabilities are. Particularly is it necessary to proceed slowly when changing a patient from regular insulin to protamine insulin or when a combination of regular insulin and protamine insulin is employed. Surely allow a week for the transfer or you will rue it. Remember that if sugar shows in the late forenoon, regular insulin may need slight increase, but if the urine is sugar-free on rising the regular insulin may require diminution. If sugar shows during the latter part of the day and particularly on rising, the protamine insulin may need an increase. Sometimes, however, sugar will show on retiring because of too great a load of carbohydrate, and because of the slow but persistent action of the protamine insulin or the mild character of the disease, the urine will become sugar-free before morning. Thus, caution should be employed in giving regular insulin on rising because with a normal blood sugar it may act so quickly that a reaction may occur even before breakfast. Indeed regular insulin, if given at the same time as protamine insulin, should be injected at a shorter interval before breakfast than if it is given alone. On the other hand, it is a comparatively indifferent matter as to the exact minute when protamine insulin is injected. The effect of the slow action of protamine insulin must be considered in the arrangement of the diet and in the treatment of reactions.

The dietitian again comes into her own diabetic sphere with the discovery of protamine insulin, because protamine insulin, powerful though it is, cannot overcome the peak carbohydrate load of diabetic diets hitherto employed and their rearrangement is essential. A greater space of time between meals becomes necessary—an earlier breakfast and a later dinner. The concentration of food into nine hours, as so often practiced in hospitals, with an eight o'clock breakfast and a five o'clock supper, is out of the question with the modern diabetic taking protamine insulin. Not only should the early and late meals be spread apart but a portion of the carbohydrate should be given during the forenoon, afternoon, and upon retiring. Only in this way can one take full advatage of this new medicine; indeed, by so doing the liability of insulin reactions becomes remote.

Insulin reactions (hypoglycemia) can occur with protamine insulin, it is true, but if the diet is distributed throughout the twenty-four hours they are far less apt to develop. Protamine insulin is a powerful, although a slowly moving force, and even if a patient comes out of a reaction one hour, he may require treatment at each of several subsequent hours. This is one of the reasons why always in the use of the protamine insulin one should not be misled by single tests of the urine and blood, but rather be guided by the general trend of these tests. Insulin reactions due to protamine insulin are generally recognized earlier by the patient and may be thwarted, if he is alert and understands how insulin and protamine insulin behave. They are most apt to develop following the use of both regular and protamine insulin, but they can occur with protamine insulin alone and, although this is seldom the case, they can be extremely violent, requiring not only large quantities of carbohydrate by mouth but intravenously as well. Here it is that knowledge helps the diabetic and to prove it you, and doctors like you, have furnished me the evidence, because more than 300 doctors have been under my care for diabetes.

Diabetic doctors obviously understand some-

thing about diabetes. By no means are they or you infallible, but none the less my diabetic doctors have demonstrated several facts.

1. Today you cannot get a diabetic doctor to die of diabetic coma! Indeed, not one death from diabetic coma has taken place among my 300 diabetic doctors during the last 14 years. If a doctor will not die of diabetic coma, why should he permit a patient to do so?

2. Up into middle age the death rate of my diabetic doctors is less than one-quarter that of my other patients of similar age.

3. The average age of death for doctors in the United States is 63 years, but for my diabetic doctors, many of whom, I will allow, contract their diabetes above the age of 40, it is 68 years.

Knowledge counts. All diabetics cannot study to be doctors, but I am convinced that the more my diabetics learn what my diabetic doctors know about diabetes, the longer they will live.

PRACTICAL POINTS IN THE ADMINISTRATION OF PROTAMINE INSULIN

I am definitely advised that protamine insulin, when combined with zinc or calcium, will not lose more than 10 per cent of its strength in 6 months, even if not kept in the ice chest. In other words, a patient taking 20 units of protamine insulin at the end of 6 months may require 22 units of the same supply. Even when the protamine and buffer have been mixed with insulin, I am likewise assured its strength will not deteriorate for upwards of 3 weeks* or for practical purposes until the contents of the bottle are exhausted. Protamine and insulin can be mixed and injected at once, but it may be a trifle better to allow the mixture to remain overnight before use. In the administration of regular and protamine insulin we have, for purposes of future study, arbitrarily adopted the plan of injecting regular insulin into the left half of the body and protamine insulin into the right half. Local reactions from protamine insulin occur just as they do with regular insulin and perhaps somewhat more frequently, but with time disappear. The efficacy of protamine insulin lies in the precipitate. Consequently, one should twist or gently turn the bottle after mixing the two solutions instead of creating a foam by shaking violently, because the particles of protamine insulin might collect in the foam. Fortunately with the newer combinations, protamine calcium insulin and protamine zinc insulin, the precipitate is more finely flocculent and uniform and less likely to adhere to the walls of the bottle. If a clear fluid is present in the bottle after mixing, it is valueless because there is no insulin in it. In the administration of regular insulin and protamine insulin the same syringe can be employed, but regular insulin should be given first. The syringe and needle can be sterilized as usual with alcohol and neither need be absolutely dry.

*I know that the protamine zinc insulin, when combined in one bottle, retains its full potency for more than 4 months, because of results obtained with eight of our standard cases.

CONCLUSIONS

1. A single injection of protamine insulin replaces multiple injections of regular insulin.

2. Due to its slow action the danger of insulin reactions is lessened.

3. Its prolonged effect, extending throughout the entire 24 hours, insures far more efficient control of the diabetes, greatly reduces the liability to coma or slowly developing complications of the disease and thus offers the diabetic almost a return to normal health.

4. The simplicity of administration of protamine insulin will appeal to the great mass of diabetics of mild and moderate degree, many of whom have needed but have not taken insulin, and thus should raise the standard of treatment of diabetes throughout the world. Perhaps this will be its greatest use.

5. Hagedorn's discovery of protamine insulin has opened up research in the treatment of diabetes along new avenues and I believe it only just and proper to name the new era thus begun after its discoverer—the Hagedorn era.

CHAIRMAN MINOT: I declare that the Nutrition Symposium is completed and the nutritious hospitality of the medical school faculty and faculty wives will be found in the courtyard.

The Massachusetts Medical Society

PROCEEDINGS OF THE COUNCIL

Special Meeting, November 19, 1936

A SPECIAL meeting of the Council of the Massachusetts Medical Society was held in John Ware Hall, Boston Medical Library, on Thursday, November 19, 1936, at 11:00 o'clock. The following 120 Councilors were present:

BARNSTABLE
M. E. Champion
S. M. Beale, Jr.
W. D. Kinney
J. I. B. Vail

BERKSHIRE
W. T. Frawley

BRISTOL NORTH
W. H. Allen
F. V. Murphy

BRISTOL SOUTH
E. F. Cody

ESSEX NORTH
E. S. Bagnall
R. V. Baketel
C. S. Benson
J. F. Burnham
Z. W. Colson
H. F. Dearborn
G. L. Richardson
W. D. Walker

ESSEX SOUTH
J. F. Jordan
C. H. Phillips
J. W. Trask

FRANKLIN
W. J. Pelletier

HAMPDEN
P. E. Gear
E. A. Knowlton
M. W. Pearson
G. L. Steele

MIDDLESEX EAST
J. H. Kerrigan
J. H. Blaisdell
Richard Dutton
E. M. Halligan
R. R. Stratton

MIDDLESEX NORTH
F. P. Murphy
A. R. Gardner
T. A. Stamas
E. O. Tabor
M. A. Tighe

MIDDLESEX SOUTH
S. H. Remick
C. F. Atwood
E. W. Barron
E. H. Bigelow
B. F. Conley
D. F. Cummings
H. F. Day
D. C. Dow
A. W. Dudley
W. G. Grandison
C. M. Hutchinson
A. A. Levi
F. P. Lowry
R. A. McCarty
L. W. McGuire
J. A. McLean
Edward Mellus
C. E. Mongan
J. P. Nelligan
E. J. O'Brien, Jr.
C. T. Porter
W. D. Reid
T. E. Reilly
E. S. A. Robinson
E. J. Sawyer
M. J. Schlesinger
E. F. Sewall
H. P. Stevens
H. W. Thayer
Fresenius Van Nüys

NORFOLK
Maurice Gerstein
F. G. Balch
H. G. Batchelder
A. S. Begg
D. D. Berlin
H. K. Boutwell
D. G. Eldridge
H. M. Emmons
I. A. Finkelstein
C. S. Francis
J. B. Hall
L. F. Johnson
W. B. Keeler
C. J. Kickham
H. M. Landesman
W. A. Lane
J. S. H. Leard
Samuel Nadel
H. C. Petterson
Frederick Reis
M. V. Safford
H. F. R. Watts

NORFOLK SOUTH
T. B. Alexander
R. L. Cook
W. G. Curtis
G. V. Higgins
F. E. Jones

PLYMOUTH
P. H. Leavitt
F. F. Weiner

SUFFOLK
Conrad Wesselhoeft
H. L. Blumgart
C. S. Butler
Lincoln Davis
R. L. DeNormandie
Reginald Fitz
Channing Frothingham
E. P. Joslin
R. I. Lee
C. C. Lund
J. P. O'Hare
R. B. Osgood
L. E. Parkins
Helen S. Pittman
R. M. Smith
M. C. Sosman
E. F. Timmins
I. J. Walker

WORCESTER
R. J. Ward
J. C. Austin
W. P. Bowers
G. A. Dix
G. E. Emery
A. W. Marsh
E. H. Trowbridge
F. H. Washburn
R. P. Watkins

Immediately after the call to order the President, Dr. Mongan, declared the meeting to be in executive session. The Council passed the following motions:

1. To approve of the action taken by the eighteen district presidents at the meeting held on July 15, 1936, in forwarding to His Excellency, the Governor, certain resolutions expressing confidence in the conduct of the Department of Mental Diseases and recommending the reappointment of the Commissioner in office at that time. It was pointed out that, when these resolutions were adopted and passed, there was no other candidate before the Council and the action was intended to assist the Governor in his choice of a Commissioner.

2. It was voted to confirm the action of the President of the Massachusetts Medical Society on November 10, 1936 asking the Governor and Council for an opportunity to be heard in the matter of the proposed appointment of a Commissioner of Mental Diseases.

3. The following resolution was passed:

> "Whereas, The statute of the Commonwealth requires that the Commissioner of Mental Diseases be a physician 'expert in the care and treatment of the insane'; therefore be it
>
> "Resolved, That the Council of the Massachusetts Medical Society direct the President to convey to the Governor and Council its conviction that the most careful consideration be given to the matter of the qualifications of any person suggested for appointment as Commissioner of Mental Diseases."

The meeting adjourned at 11:45 a. m.

ALEXANDER S. BEGG, *Secretary.*

URINARY FISTULAS OPENING INTO THE VAGINA*

BY FRANK A. PEMBERTON, M.D.,† GEORGE VAN S. SMITH, M.D.,† AND SIDNEY C. GRAVES, M.D.†

ONE hundred cases of urinary fistula have been treated at the Free Hospital for Women in Brookline between 1879 and June, 1936; 57 of these have been seen since 1915.

The fistulas of 61 cases were of obstetrical origin; those of 28 were caused by some operative procedure other than for cancer; the remaining 11 were due to cancer or its treatment by operation or irradiation. The types and combinations of fistulas for the whole group are listed as follows:

Vesicovaginal	75	Urethrovaginal	3
Double vesicovaginal	5	Double urethrovaginal	1
Triple vesicovaginal	1	Vesicocervicovaginal	12
Vesico- and urethrovaginal	1	Ureterovaginal	2

In addition, 4 cases had vaginal atresia, of which 1 had also a complete laceration of the perineum. Another case had a complete perineal tear. A cancer case had also a rectovaginal fistula. One patient's posterior urethra was completely occluded by scar tissue. In another patient was a draining wound from suprapubic cystotomy. The distal three-fourths of the urethrovaginal septum was found torn in one case. Another patient's distal one-third of the urethrovaginal septum was split open, this and her fistula having followed a plastic operation at home. A large proportion of all the cases had scarring of greater or less degree. Furthermore, local incrustation, cystitis, pyelitis and contracted bladder were not uncommon. Although this report deals chiefly with the closure of fistulas, these associated complications and lesions necessitated both medical treatment and other plastic procedures in addition to those aimed at the repair of fistulas.

As to size, 58 per cent of the fistulas were 2 millimeters or less in diameter; 34 per cent were 3 to 10 millimeters in diameter; 4 per cent were about 15 millimeters in diameter; and 4 per cent were over 2 centimeters in diameter, the largest of the series being 6 centimeters (a recent case, now dry 6 months after one operation).

Sixty-three per cent of the patients whose fistulas followed delivery were primiparous. The data concerning delivery for the entire group with fistulas of obstetric origin are given in table 1. Table 2 summarizes the surgical procedures for the 28 cases whose fistulas followed operation performed for nonmalignant conditions. Eight of these acquired their fistulas at the Free Hospital for Women before 1906—1 from a vaginal plastic, 1 from a complete hysterectomy and 6 from intentional incision as part of the one-time treatment of cystitis.

TABLE 1

DELIVERY NOTES ON 61 CASES OF URINARY FISTULA OF OBSTETRIC ORIGIN

Instrumental (type not stated)	34	(Stillborn—8)
High forceps	6	
Long labor; e.g., 2, 3, 3 and 8 days	6	(Stillborn—2)
Indefinite	10	(Stillborn—7)
Normal	1	(Stillborn)
Breech	1	
"Posterior position"	1	
Face presentation	1	
Vaginal hysterotomy	1	

Total stillborn—18, 29.5 per cent

TABLE 2

PROCEDURES PRECEDING THE DEVELOPMENT OF URINARY FISTULA IN 28 CASES

Complete abdominal hysterectomy	9
Supravaginal hysterectomy	3
Abdominal myomectomy	1
Vaginal plastic	6
Vaginal drainage of postoperative abscess	1
Operation for bladder calculus	1
Insertion of cup-shaped pessary	1
Intentional fistula for treatment of cystitis	6

In considering fistulas associated with pelvic cancer (almost always cervical) it is difficult to determine which are due to the disease and which to its treatment. They are quite common in advanced cancer of the cervix. Actually at this clinic to date 21 urinary fistulas in cancer cases are considered the result of treatment. Eleven fistulas in cancer cases are included in this report because they were repaired. Seven resulted from radical hysterectomy, 2 from irradiation and 2 from the disease.

Spontaneous closure. Now and then fistulas close spontaneously after the clearing up of a bladder infection or the removal of a foreign body or during the healing process following the operation that caused the opening. One patient reported herself cured 17 years after irrigations which removed much calcareous material from the bladder and vagina in preparation for operation on a vesicocervicovaginal fistula. She left the country before operation and has not been examined since.

Another patient's vesicovaginal fistula, 1 millimeter in diameter, closed 9 months following su-

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prapubic cystotomy for the removal of a calculus 2.5 centimeters in greatest diameter. She was 23 years old on admission. At the age of 14 she had been under treatment for 6 months because of urinary frequency with burning and pain. A diagnosis of renal tuberculosis had been made. During this period incontinence developed. It persisted for 1 year and then ceased. She was then well until the age of 20 when, she related, seven stones were passed in 3 days. Following this episode her course had been uneventful until she became pregnant 10 months before admission. Incontinence ensued until she miscarried at 4 months. Two weeks later she experienced much pain and hematuria with the passage of sand. Six weeks before entry incontinence recurred associated with almost constant pain. Genito-urinary studies revealed fistula, calculus and bilateral renal tuberculosis with calcification, more marked on the right. By x-ray the lungs showed no evidence of tuberculosis. Four and one-half months after cystotomy the right kidney and upper ureter were removed. Convalescence was slow and the patient was not discharged until 3 months after nephrectomy. After a short stay at a sanatorium she left against advice. Two years and one month after cystotomy she was symptomatically well.

Three vesicovaginal fistulas healed without interference 3, 5 and 7 months, respectively, after radical operations for cancer of the cervix. A left ureterovaginal fistula following a complete hysterectomy closed spontaneously 8 months later, at which time an intravenous pyelogram was normal. Ureteral trauma was not suspected at operation. Leakage began within 10 days. Presumably the ureter had been caught in a suture.

These 6 cases constitute this clinic's experience with spontaneous closure, but are not included in the series because repair was not performed. It is interesting to note that of the 16 failures in the series (2 were operative deaths), 9 were followed for 4 months, 9 months, 1 year and 4 months, 1 year and 9 months, 3 years, 3 years, 6 years, 9 and 30 years, respectively, and spontaneous closure did not occur. If this fortunate outcome is to take place, evidence of less leakage, such as pads moist instead of soaked, no leakage unless bladder fills beyond a certain capacity and leakage only on lying, standing, sitting or walking, will probably appear rather early, as indicated by the cases in which closure did come about without interference.

Bladder calculus. Occasionally treatment is complicated by the presence or development of a stone. With the exception of the above instance, a calculus has not been found in a previously untreated case. The possible causes are failure to remove entirely nonabsorbable sutures, infection, stasis, alkaline urine, vitamin deficiency and endocrine dysfunction. The 3 cases of bladder calculus in this series are as follows:

CASE 1. Mrs. M. D., aged 32 years, 6 years before admission had had a vesicovaginal fistula which followed a high forceps delivery. This was repaired at another clinic 6 months later and the patient was well until a year before admission, having had a second (normal) delivery 2 years after the first and a hemorrhoidectomy. Her symptoms were frequency and difficulty in voiding. She had to "press on the bladder" from below to void. Two months before entry leakage recurred. A 3 centimeter calculus was removed through a vesicovaginal fistula of almost the same size. In addition, two silver wire sutures were excised when the edges of the opening were trimmed. The fistula became rapidly smaller but the outcome is unknown, since the patient ceased reporting. Though not likely, the sinus may have closed spontaneously.

CASE 2. Mrs. E. S., aged 32 years, was admitted because of urinary incontinence 4 months after the delivery of a stillborn infant. She had a complete laceration of the perineum, scarring of the upper fourth of the vagina and a 2 millimeter vesicovaginal fistula whose inner end was close to the opening of the left ureter. The fistula was closed with silver wire, great difficulty being encountered because of excessive and intractable scarring. Four weeks later the sutures were removed, the suture count being correct, and the perineum repaired. In 31 days the patient was discharged cured. One year and five months later she returned with a story of having had painful urination with the passage of gravel and blood 7 weeks before. Cystoscopy, though indicated, was not done. Another fistula was present. It recurred soon after repair and was again closed, only to appear within a month. Cystoscopy then revealed a stone. Shortly afterward a fourth attempt at repair was made, at which operation a 3 millimeter calculus was taken from the bladder through the sinus. This and a fifth operation were unsuccessful. This case is listed below among the failures, despite the fact that it seemed likely that the severe scarring and the development of stones, rather than any flaw in technic, contributed chiefly to the unsatisfactory result.

CASE 3. This patient, Mrs. B. B., aged 29 years, had had during the previous year three operations for a vesicovaginal fistula of obstetric origin. The first had been a transvesical repair through a suprapubic cystotomy, the second a hysterectomy with abdominal closure of both fistula and cystotomy and the third a vaginal repair of the fistula. Examination disclosed practically a complete laceration of the perineum, a relaxed and patulous urethra, much scarring at the top of the vagina, absence of the cervix and a vesicovaginal fistula, 5 millimeters in diameter, high up. The fistula was closed and the urethra tightened. Six weeks later, 4 days following the removal of sutures, incontinence recurred. The patient was not seen again for 5 years, during which period she had had eight reentries at another hospital with final closure of the fistula, although incontinence continued because of an incompetent urethra. Ten months before her return suprapubic cystotomy had been performed for the removal of a calculus 4 by 2 by 2 centimeters, and the patient stated that during recent months she had passed blood, shreds of tissue and several small stones. On readmission a sloughing ulcerated area was seen in the region of the left ureteral orifice, which could not be visualized or catheterized. The surface of the area was soft and crumbly, but the material removed for biopsy was amorphous. The introduction of a sound did not give the feeling of a stone. It was thought to be tumor. The right kidney and ureter were normal. Transplantation of this ureter into the sigmoid was performed in anticipation of cystectomy. The left kidney could not be felt be-

cause of adhesions. Vaginal leakage ceased completely following this procedure. Intravenous pyelography demonstrated normal function of the right and no function of the left kidney. At the next operation the vesicovaginal septum was opened and a stone one-half inch in diameter, previously thought to be tumor, was taken from the region of the left ureteral opening. Two months later the left kidney and part of its ureter were removed for hydronephrosis and hydroureter. The patient's last operation, 15 months from the time of nephrectomy, consisted of a perineorrhaphy because of anal incompetence. At her latest examination, 10 years after her first admission and 3 years following perineal repair, her seventeenth operation, she was well with the exception of occasional slight anal incontinence. Her superfluous bladder with its opening into the vagina was giving no symptoms. This case also is included among the failures, but it is likely that the patient would have been cured sooner and undergone less suffering had the early attempts at closure been undertaken by the vaginal route.

Operative technic. By the Sims' operation is meant a simple oval denudation by the vaginal approach which extends to the bladder mucosa. The edges are trimmed to permit approximation without interference by epithelium. Scar tissue is excised or mobilized as much as possible. Accurate approximation without tension is the important aim. One should not fear making the opening larger during this procedure, for it does no harm. Fistulas of small caliber are more likely to heal if considerably enlarged, probably because the dissection is certain to remove all scarred and inflamed tissue. In placing interrupted sutures of silver wire, all layers of the vesicovaginal septum except the bladder mucosa are included.

In cases where the loss of tissue and scarring are greater and where approximation is more difficult, an incision is carried around the opening, and the bladder and vaginal walls are separated from each other until the edges of the bladder opening can be drawn together without tension, using a fine tanned catgut suture. The vaginal opening is closed with a row of interrupted sutures. This is essentially the Braquehaye method except that in the latter the sinus is dissected out in a collarette and inverted into the bladder.

If the fistula is close to a ureter the introduction of a catheter into the ureter is a great help in placing the sutures so that they will not include the ureteral wall. The needle is inserted with much more confidence and there is no skimping of the bite on one side for fear that it may include the ureter.

It is sometimes necessary to resort to plastic manipulations such as rotating a flap of vaginal mucosa to fill in a defect. Rarely, the location of the sinus makes closure by the abdominal route more feasible. In this series the following types of operation were performed: Sims—99, Braquehaye—24, plastic—11, abdominal repair—2, implantation of left ureter into bladder—1, and transplantation of right ureter into sigmoid—1. This made a total of 138 operations, of which 38 were repeats. Ureteral implantation into the bladder was done for ureterovaginal fistula following complete hysterectomy for cancer of the cervix. Convalescence was normal and the patient had had no reoccurrence at death, 4 years and 6 months after implantation. Ureteral transplantation into the sigmoid was performed recently for a right ureterovaginal fistula. Convalescence was satisfactory and the patient discharged cured. She had undergone radical hysterectomy in November, 1935, for an early carcinoma of the cervix complicated by chronic pelvic inflammation with a 15 centimeter simple serous cyst of the left ovary. Following hysterectomy an abscess developed in the right side of the pelvis. This discharged into the bladder. Soon afterward, vaginal leakage of urine occurred. Scarring made implantation of the ureter into the bladder impossible.

Although advised by Sims and frequently mentioned in the literature, the use of silver wire in the repair of urinary fistulas is often ignored. At this clinic it is considered one of the most important factors in achieving a cure. The wire is placed in interrupted sutures and includes a good bite of all tissues to the bladder mucosa. It gives good support, does not tend to cut through and causes little or no irritation. Some have thought that it may form an antiseptic silver salt on contact with tissue juices and urine. It is left in for at least 3 and usually 4 to 6 weeks. At present silver-plated copper wire (no. 26) is employed. It is placed by the use of curved needles of suitable size carrying white braided silk leaders (nos. 8 to 11). The tissues are approximated without constriction by twisting the wire on itself. The ends are cut and shotted with lead. A record is kept of the number of sutures used and this number should be accounted for at the time of removal. The importance of removing all silver wire is suggested by the first of the bladder calculus cases in which retained silver wire was found associated with stone and a new fistula. It is also demonstrated by the following résumé of a case in this series:

Mrs. E. E. H. on admission had already undergone two attempts at repair. A third repair seemed successful and her vagina was dry 8 weeks after operation, when nine and one-half, instead of ten, silver stitches were removed. Two and one-half years later she returned because of constant slight leakage since discharge. A 0.5 millimeter sinus was found near the upper end of the vaginal scar and just above the scar was a 2 millimeter fistula in which was attached the other half stitch. She has now been cured for over 8 years since the fourth operation, which probably should not have been necessary.

The desideratum of postoperative care is to avoid tension on the sutures. Constant drain-

age is maintained either by a plain tipped soft rubber catheter held by adhesive strips or by a small indwelling silver catheter. These should be watched constantly to make certain they do not become plugged. The bowels are kept open. After the third postoperative day gentle douches of sterile boric acid solution are given. The catheter is removed between the ninth and fourteenth days and the patient allowed up after the fourteenth day. Removal of the sutures is carried out in the operating room with anesthesia, unless they can be easily exposed.

Although the technic of repairing a fistula is often not difficult, the result may be unsatisfactory due to incomplete knowledge of the exact type and location of the sinus, the unsuspected presence of bladder calculus, improper sutures, inadequate postoperative care in preventing sepsis and bladder distention and failure to remove entirely certain nonabsorbable suture material, such as silver wire. It is extremely important that thorough genitourinary studies be carried out as far as possible before treatment.

There were two operative deaths. The first patient, aged 53 years, had had a complete hysterectomy for cancer of the cervix in April, 1915. A fistula was unsuccessfully repaired in January, 1916. She died of pyonephrosis in May, 1916, 58 days after a second operation with satisfactory closure. The second patient, aged 35 years, had had an unsuccessful repair of her vesicovaginal fistula 6 months before admission. A second repair from below at this clinic was of no benefit. In April, 1931, an attempt to close the sinus by the abdominal route was a failure. There was excessive scarring and the approach was difficult. She died in November, 1931, during a second attempt at closure abdominally.

Results. Despite conscientious efforts to obtain complete follow-up data, there is no further information on 32 cases which were cured at the time of final discharge more than 1 year ago. The majority of these were treated before 1916. Forty-five other cases, however, cured on discharge and followed from 1 to 30 years, remained cured. The second case in the bladder calculus group developed a new fistula between 12 and 16 months after being discharged cured. This is the only known instance in the series of a change in the discharge result. From this evidence it seems fair to consider that the majority of those cured on discharge stay cured. In cases of operative failure, leakage invariably recurs within 10 weeks.

Division I. Twenty-eight patients had undergone one to seven previous attempts at closure before admission—in all 61 operations, of which 4 were suprapubic (average = 2.2 operations per case). These patients had 40 more operations at this clinic. Unsuccessful repairs tend to augment not only the difficulty of later operation but also the chances of a poor result, because of an increase in scar tissue and tension. However, as will be pointed out below, failures should not cause despair, since success so frequently follows repeated attempts. The results in this group are summarized as follows:

Operative death during third attempt (abdominally)	1
Cured on discharge	6
Cured one to two years later	2
Cured two to fifteen years later	9
Fistula cured but functional incontinence two and six years later	2
Cured three, eight and thirteen years after second operation	3
Fistula cured but urethra incompetent six years after second operation	1
Fistula closed but urethra incompetent two years after third operation	1
Failures—no further operation	2
Failure (third case under bladder calculus)	1
Primary cures 19, 67.8 per cent	
Final cures 24, 85.7 per cent	

One patient had one normal delivery subsequently and later gave birth to premature twins without complication. Another had three normal deliveries without further mishap.

Division II. Sixty-one cases untreated before admission underwent 85 operations (average = 1.38 operations per case). The results in this group are summarized as follows:

Cured on discharge	24
Cured one to two years later	4
Cured two to over ten years later	17
Cured on discharge after second operation	2
Cured two years after second operation	1
Cured eighteen years after third operation	1
Cured on discharge after fourth operation	2
Cured on discharge after sixth operation	1
Failures—no further operation	5
Failures—after two attempts	2
Failure—after three attempts	1
Failure—after five attempts (second calculus case)	1
Primary cures 45, 73.7 per cent	
Final cures 52, 85.2 per cent	

Three patients had one normal delivery each and one had two deliveries without complication. A fourth patient had one miscarriage and one premature birth; a fifth, one miscarriage.

Division III. The results in 11 cases with fistulas caused by cancer of the cervix or its treatment are as follows:

Operative death following second attempt	1
Cured on discharge	3
Cured until death two years later	1
Cured until death four years and six months later	1
Cured fifteen years later	1
Recurrence due to disease three months later (1892)	1
Cured until death two years after second operation	1
Failures	2
Primary cures 7, 63.6 per cent	
Final cures 8, 72.7 per cent	

Thus, of 100 treated cases, 71 were cured by one operation at this clinic and 84 were final cures. None of the surviving uncured patients were made worse by treatment. On the contrary, 2 were markedly improved. There were four instances in which urinary incontinence persisted to an annoying degree even after satisfactory healing of the fistula. It is not unlikely that the mechanism of sphincter control may have been upset from previous trauma, scarring and lack of use, for in these cases the urethra was not unduly patulous and, furthermore, plastic operations which ordinarily benefit functional incontinence were of little help. Six other patients had varying degrees of functional incontinence but none seemed sufficiently incommoded to demand repair. The insertion of a pessary completely relieved 1 of these. Another patient reported complete control within a few months after operation. A third regained complete control by the end of the second postoperative year.

Discussion. In general it would seem wise not to attempt repair of fistulas as soon as they have been incurred. An occasional one will heal spontaneously, especially if there is some evidence of retention. Fistulas should not be repaired while the patient is nursing, because the so-called lactation atrophy of the genital organs, with their reduced blood supply, may not be conducive toward healing. Failures may be due to eagerness on the part of the surgeon to relieve the patient from her distressing condition instead of waiting for a favorable time to operate. In cases of fistula due to irradiation, operation should not be undertaken until the tissues have had plenty of time to recover from the effects of treatment—at least a year after the development of the sinus.

Although there has been no experience at this clinic with the transvesical repair of fistulas through a suprapubic cystotomy, the risks and disadvantages of this procedure are considered too great to warrant its use. Rarely one is compelled to operate transperitoneally, as happened in one of the above cases in which the fistula opened high up in the endocervix and in another in which the depth of the vagina and the scarring made approach from below technically impossible. Operations from below incur no great risk and convalescence tends to run smoothly.

There seems to be an increasing trend, especially among urologists, toward transplanting the ureters into the intestine in cases of vesicovaginal fistulas with large openings or of those apparently inaccessible ones located at the top of the vagina, as after complete hysterectomy. No attempt at repair should be made until all induration has gone and the tissues are as flexible as they are going to be. Then it is not uncommon to find that the bladder wall can be drawn down with comparative ease and a direct attack made. No definite rules can be laid down regarding transplantation of the ureters but, if the fistula can be reached and its edges drawn together through the vaginal approach, the much more dangerous operation is unnecessary.

Since 13 cases were finally cured after two to six operations, one should not become discouraged if one attempt results in failure.

SUMMARY

One hundred treated cases of urinary fistula opening into the vagina have been reviewed as to type, size, cause and complicating factors.

Cases of spontaneous closure and those with bladder calculus have been cited.

The management, operative technic, errors and causes of failure have been considered and the results presented. Final cures were 84 per cent.

MOULDED PLASTER RIGHT ANGLE ELBOW SPLINTS

BY PAUL R. WITHINGTON, M.D.*

THOSE who have used anteroposterior moulded plaster splints for the immobilization of Colles' fractures know how satisfactory they are. They may be applied without danger of subsequent slipping, are comfortable, and, since they conform to the soft parts, are unlikely to produce pressure sores. However, when the fracture is of the middle or upper third of the forearm, it is, of course, necessary to immobilize the elbow joint. To do this one must, in addition to the anteroposterior splints, use an internal angular splint of tin. Not only does this make a bulky dressing, but it also requires that the doctor have on hand a large collection of different sized splints. The alternative is to apply a circular plaster of Paris splint. In the application of this, one is apt to lose a previous good reduction due to the repeated shifting of the bandage from hand to hand. Another disadvantage is that the cast may have to be split (if swelling occurs) or renewed (if, as swelling goes down, mobility of the fragments occurs). A strip of plaster laid out at right angles will not, when applied to the elbow, hold the joint motionless because of the play allowed by the soft tissues about the upper arm.

In order to obtain the advantages of the moulded plaster splints for this type of frac-

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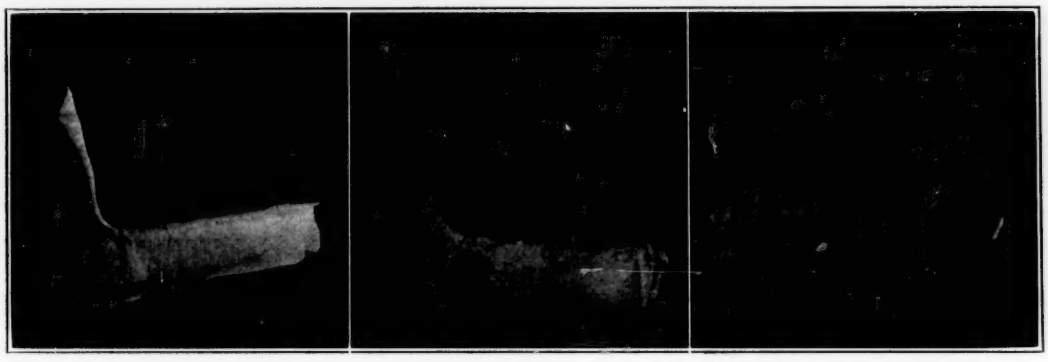
ture, and at the same time to offset the disadvantage of the superimposed internal angular splint, it occurred to me that if, instead of laying the plaster out at right angles, it were laid out at a slightly acute angle (for the anterior splint) and at a slightly obtuse angle (for the posterior one), the ends would, on application, curl around to the anterior and posterior respectively, of the upper arm, thus holding the elbow firm. This utilization of the third dimension solved the problem; the splint here pictured, resulted.

The advantages of the splints are as follows:

1. Lightness and compactness.
2. Less chance of losing a good position due to shifting of hands during application.
3. Less chance of pressure sores; ease of accommodation to increase or diminution of swelling of the arm.
4. Ease in the dressing of compound fractures.
5. Does away with the need of a large assortment of tin internal angular splints.
6. Toward convalescence the splints may easily be trimmed to a smaller size.



Anterior splint, Rt. (internal view).

Posterior splint, Rt. (external view).

Both splints, arranged as though in position (from above).

COURT DECISION ON PUBLIC HEALTH

Tuberculosis, contracted by employee in business of manufacturing women's dresses because of conditions of employment, held not compensable as occupational disease under workmen's compensation act. —(Connecticut Supreme Court of Errors; Madeo v. I. Dibner & Bro., Inc., et al., 186 A. 616; decided July 30, 1936.) An employee in the business of manufacturing women's dresses claimed compensation under the Workmen's Compensation Act for disability due to pulmonary tuberculosis. The finding disclosed that the tuberculosis from which she suffered was contracted because of conditions of employment. The commissioner awarded compensation, but the trial court sustained the appeal of the defendants, and the plaintiff appealed to the supreme court of errors.

The compensation act defined a personal injury as including "occupational disease", which, in turn, was defined as "a disease peculiar to the occupation in which the employee was engaged and due to causes in excess of the ordinary hazards of employment as such."

The supreme court quoted from a prior case in which it had said that "to come within the definition, an occupational disease must be a disease which is a natural incident of a particular occupation, and must attach to that occupation a hazard which distinguishes it from the usual run of occupations and is in excess of that attending employment in general." Regarding this definition, the court, in the instant opinion, stated that "It does not include a disease which results from the peculiar conditions surrounding the employment of the claimant in a kind of work which would not from its nature be more likely to cause it than would other kinds of employment carried on under the same conditions." "In this case", said the court, "the plaintiff's disease resulted from the conditions of her particular employment in the factory of the defendants. Other trades carried on under those conditions would have been as likely to cause the disease as the manufacture of dresses."

The action of the trial court in denying compensation was sustained.—*Public Health Reports*, October 30, 1936.

FOOD AND DRUG PENALTIES COVER VARIETY OF OFFENSES

Fines imposed in cases brought under the Federal Food and Drugs Act during October ranged from $1 and $2 up to $300 and $500, the Food and Drug Administration reports. The total of fines assessed was $2,563.

Criminal prosecutions were based on decomposed or otherwise questionable foods, and a variety of fraudulent preparations of drugs.—*U. S. Department of Agriculture.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 22511

PRESENTATION OF CASE

A 22 year old college girl was admitted complaining of pain in the abdomen, and backache.

The patient had been well until the day before entry when, following a large noonday meal, she developed vague upper abdominal pain and backache. The pain was not severe and was not associated with nausea or vomiting. That evening her appetite was poor and she ate but little for supper. She took a cathartic shortly before going to bed and subsequently had four bowel movements between 2 and 4 a. m. She slept poorly and her abdominal discomfort during the night shifted to the upper abdomen and both lower quadrants of the abdomen. On arising on the day of entry the patient had a light breakfast following which she was nauseated and vomited once. She went to her first class but had sufficient malaise so that she then went back to bed. The patient felt slightly feverish but there was no further vomiting or diarrhea. Her backache was not the same in character as she occasionally had with her menses. It was not localized to one area but extended generally across her back below her twelfth ribs. Equal discomfort was experienced on both sides. Her abdominal discomfort persisted with much the same intensity as when first noted.

The menses had begun at the age of 13 and were regular every 28 days with a duration of 6 days. The last period was 2 weeks prior to entry. There had been no previous illness of significance or any attack similar to the present illness.

Physical examination showed a well-developed and nourished young girl lying quietly in bed in only slight discomfort. Save for her abdominal and pelvic findings, no abnormalities were noted on general examination. Her abdomen was not distended. Peristalsis was somewhat diminished but when heard was normal in quality and pitch. Liver dulness was not obliterated and the liver edge could not be felt. On palpation there was some tenderness in the midline just above the umbilicus, extending somewhat into the right upper quadrant. There was also slight tenderness in the right lower quadrant. There was but slight involuntary muscle spasm associated with the tenderness in these two areas. The left lower quadrant and the left upper quadrant were not tender. The tenderness in the upper part of the abdomen was definitely more marked than that in the right lower quadrant. No well-defined regional tenderness was elicited. Pelvic examination showed the pelvic organs to be normal in size and position. There was definite but slight tenderness in the left vault. No mass was felt. No leukorrhea was present. Rectal examination added nothing further to the pelvic findings.

The temperature was 100°, the pulse 120. The respirations were 20.

Examination of the urine showed a specific gravity of 1.020. There was no albumin or sugar, but a positive reaction for diacetic acid and acetone was obtained. A later urine examination was negative. Examination of the sediment was negative. The blood showed a white cell count of 15,500, 85 per cent polymorphonuclears.

A flat x-ray plate of the abdomen showed no abnormalities.

Shortly after entry a laparotomy was performed. A noninflamed appendix folded at the junction of its distal and middle thirds was exposed. This thickened area of the appendix was twice the size of a normal appendix and was firm in consistency. Further exploration showed a mass the size of a golf ball in the midline retroperitoneally, just below the transverse mesocolon.

DIFFERENTIAL DIAGNOSIS

DR. ROBERT R. LINTON: I think it is fair to say that, from the history as it is given us here, we are dealing with what may be called an acute surgical abdomen. Certainly when anyone of this age presents such a story beginning with vague upper abdominal pain which shifts to the right lower quadrant, followed by nausea and vomiting, and which is associated with a slight amount of temperature, an elevated pulse and a white cell count of 15,500 with 85 per cent polymorphonuclears, the most likely thing we think of is acute appendicitis. I would hazard a guess that that was the preoperative diagnosis. However, there are certain things which are not usual in this story and one of these is the fact that she complained of backache. I think that is very unusual in appendicitis. Apparently it was present on both sides of her back. The other thing that is a little unusual for appendicitis is the very bizarre arrangement of the tenderness in the various quadrants as it is described here. I am a little hazy as to exactly where the maximum

tenderness was, but I will assume that it was a little above the umbilicus and a little to the right. That is unusual in appendicitis but it certainly could occur there.

I presume the x-ray was taken to rule out any possibility of kidney stone, as that probably was considered because of the pain in the back. I think the facts that she had no urinary symptoms, that the urine was negative, and that the x-ray plate showed no shadows, rule out her genito-urinary tract as a cause of her symptoms.

I became more confused as I read the description of the appendix. I am a little bit in doubt as to why I was given this additional information about the appendix.

Dr. Tracy B. Mallory: I thought I would like to put the question up to you exactly as it was put to the surgeon at 2 a. m. He resected this thickened appendix, opened it, and found the distal two-thirds completely solid. He felt a mass retroperitoneally. What was he dealing with? And what was he to do? There was no pathologist available at 2 a. m. to tell him what to do!

Dr. Linton: There are other possibilities which must now be considered rather than acute appendicitis. In the first place, there is a condition which is called carcinoid which, as I understand it, is a tumor-like condition of the appendix which usually involves the distal end, and which on gross and microscopic examination suggests a carcinoma. The strange thing about it, as I understand it, is that, in spite of its microscopic similarity to carcinoma, it never metastasizes. It is a relatively benign growth and whether it spreads locally or not, I am not very sure; but at least I do know that it is not supposed to metastasize.

Another possibility is that we might have a true carcinoma of the appendix. I think that is a very rare condition and for that reason alone I would rule it out as a probability, despite the fact that she had this mass in the retroperitoneal region.

There is another condition that is called mucocele of the appendix which usually involves the distal portion. It is not a solid tumor, but a cyst containing mucus. It may or may not be locally malignant. There are two other conditions which are infectious in nature which one might think of. One is tuberculosis and the other actinomycosis. It is fair to say concerning these two that the cecum is primarily involved and the appendix only involved secondarily to the cecum. The cecum was not involved in this case so I think we can rule out these two conditions. I feel that the pain in her back was explained undoubtedly by the finding of a mass in the retroperitoneal region. Just what connection that plays with this enlarged appendix, I am not very sure. I feel that we can say that it is probably a retroperitoneal gland that is enlarged due to some infection, and I am going to assume that that infection is in the appendix. I think she does not have an acute appendicitis although there may be more or less subacute infection associated with the process she has there.

There is one other thing I did think of which I wish to mention, and that is Hodgkin's disease. I think the surgeon did right in removing the appendix, even though it was Hodgkin's disease. My final diagnosis is carcinoid of the appendix with mesenteric adenitis.

Preoperative Diagnosis

Acute appendicitis.
Ruptured follicular cyst?

Dr. Robert R. Linton's Diagnosis

Carcinoid of the appendix with mesenteric adenitis.

Pathologic Diagnosis

Carcinoid of the appendix with metastasis to a retroperitoneal lymph node.

Pathologic Discussion

Dr. Mallory: I am sorry Dr. Faxon is not here to tell you his own impressions in the case. He first removed the obviously abnormal appendix and then, as I said, cut into it and proved to his own satisfaction that there was a firm tumor in the distal portion. He next attempted to explore this retroperitoneal mass a little further. He put a needle into it but was unable to extract anything. He considered the possibility of dissecting it out without knowing what it was, but since it was in intimate contact with the inferior mesenteric artery he thought that was a good deal of risk to take, not knowing what he was dealing with, so he eventually took a small biopsy and then backed out.

The histologic sections of the appendix showed a typical carcinoid and the biopsy from this retroperitoneal node showed the same thing. Carcinoids are tumors of relatively very low malignancy but they are not entirely benign tumors. It is quite true that carcinoids in the appendix, the commonest site for them, very rarely metastasize, but carcinoids also occur in the lower ileum and those not so infrequently do metastasize. We have had three ileac carcinoids with frank metastases and we have had nearly a dozen with marked deep invasion of the wall of the ileum. Even when these tumors do metastasize their growth is very slow. We have one particularly interesting case of primary carcinoid of the ileum which Dr. Daniel Jones resected. The man died 15 years later of some entirely different condition, yet at postmortem examination we found a lymph node

containing carcinoid in the retroperitoneal tissues. Metastasis had occurred but it had not spread beyond that single lymph node in the course of 15 years. On that basis Dr. Faxon decided that it was worth while to go back again in this case, which he did the day before yesterday, and was able to remove the single enlarged gland without much difficulty. I think the possibility of cure in this case is fairly good, and even if she is not cured I expect she will have a number of years of good health.

A PHYSICIAN: Do they respond to radiation?

DR. MALLORY: I do not believe anybody knows. They are sufficiently uncommon, and metastasis is so rare that I do not believe anyone has, wittingly, ever treated metastatic carcinoid.

A PHYSICIAN: How do you correlate the clinical finding with the finding at operation.

DR. MALLORY: I cannot explain all the shifts of pain. I would be inclined to agree with Dr. Linton that the back pain was due to the retroperitoneal mass. We know that tumors of the pancreas very commonly produce back pain and I think that was the one lead in the clinical history possibly pointing to this diagnosis.

DR. THOMAS V. URMY: How do you explain the acute symptoms?

DR. MALLORY: I have no answer for that.

A PHYSICIAN: Do you think it was infectious?

DR. MALLORY: No.

A PHYSICIAN: How do you recognize a carcinoid?

DR. MALLORY: That is hard to answer offhand. To anyone who has seen carcinoids, one glance with low power tells you it is carcinoid and not carcinoma. The cellular pattern is quite different. If you want more than that, you have to resort to elaborate technical methods. Masson has proved that carcinoids are not tumors of epithelial cells but tumors of nerve end-organs in the lower intestinal tract and that they contain cells which react with silver; in other words, they are argentaffin-cell not epithelial tumors.

CASE 22512

PRESENTATION OF CASE

A 24 year old unmarried American stenographer was admitted complaining of pain in the abdomen.

The patient had been perfectly well until 3 days before entry, when she was awakened at 5:00 a. m. by severe lower abdominal pain which was steady in character. She was given an enema which caused the pain to become worse and she fainted. After returning the enema, however, she felt better although there was residual steady pain in the right lower quadrant and some tenderness persisted without abatement until admission. There were no chills, nausea or vomiting. She had vomited twice about a week before entry after an evening meal, but there was no associated nausea or malaise at that time.

Five or six years ago there had been an attack of lower abdominal pain almost as severe as the current illness. During the interval, however, there had been no discomfort. The catamenia was said to have been irregular in that occasional amenorrhea occurred for 2 or 3 months. There was no menorrhagia, metrorrhagia or dysmenorrhea. The last menstrual period began 9 days before admission and lasted 5 days.

Physical examination showed a well-developed and nourished young woman in no apparent discomfort. The heart and lungs were normal. The lower abdomen was slightly distended and there was moderate tenderness and spasm in the right lower quadrant. Peristaltic sounds were normal and there were no palpable masses or costovertebral tenderness. Pelvic examination showed the fundus to be normal in size and position. It was freely and painlessly movable. Neither vault was tender but rectal examination revealed tenderness on both sides.

The temperature was 99°, the pulse 102. The respirations were 24.

Examination of the urine was negative. The blood showed a white cell count of 17,000.

Shortly after entry a laparotomy was performed.

DIFFERENTIAL DIAGNOSIS

DR. HORATIO ROGERS: This sudden onset in a young woman who was previously well, with a story of 3 days of abdominal pain in the right lower quadrant, would make one think first of appendicitis. That is by far the commonest cause of pain in the right lower quadrant. Secondly, one would think of an ectopic pregnancy. Next would come twisted ovarian cyst, ruptured follicular cyst, and possibly acute salpingitis or chronic pelvic inflammation, in about that order of likelihood. The picture does not exactly fit any of these diagnoses, however. There are several hints that would lead us to consider ectopic pregnancy. For instance, the fact that she fainted in the first attack of sudden pain. It was sudden because she woke up with the pain, having felt all right the previous evening. She had vomited twice the week before for no apparent reason, possibly a suggestion that she may have been in the early stages of pregnancy. Her catamenia is said to have been irregular in the past but it is stated with authority that her last period started 6 days before the onset of symptoms and had just ended the day before. But we cannot be sure that that was really her period. It may have been a little abnormal

uterine bleeding accompanying an ectopic pregnancy. No mass was felt on pelvic examination in either vault. The uterus is stated to have been normal in size; that is, normal for a nonpregnant uterus, and in an ectopic, of course, the uterus would be the same size that it would be in an intrauterine pregnancy of the same duration. I think that those are points which we cannot twist around, and on that evidence I do not think that we can make a diagnosis of extrauterine pregnancy.

As to acute salpingitis or pelvic inflammation, there is very little to go on and there is one fact which absolutely rules them out. The uterus was freely movable, with no pain and no tenderness in either vault on vaginal examination.

A ruptured follicular cyst has a sudden onset with pelvic tenderness. There is no vomiting here, which she would have been likely to have if it had been a ruptured follicular cyst. Pain and tenderness persisted for three days in her right lower quadrant and when she came into the hospital there was spasm on the right but not on the left. I think we have to visualize what was going on for the 3 days before she entered the hospital. In that time we would expect blood to have spread across the pelvis and the symptoms to be bilateral instead of unilateral, as far as pain and tenderness are concerned. But in this girl all the symptoms stayed on the right side for 3 days. What the rectal tenderness on both sides was, I do not know, but I am hoping it was not very important and may not be an accurate observation. Furthermore, ruptured follicular cyst should have happened halfway between her periods. This episode happened 6 days after the onset of her last period, or just 8 days too soon for the usual ruptured follicular cyst. If she was ovulating in a normal rhythm, as presumably she was if she was menstruating in a normal rhythm, then one would probably be wrong in diagnosing follicular cyst.

As regards twisted ovarian cyst, there was a sudden onset but there is not enough evidence to make the diagnosis on pelvic or on abdominal examination. She did not seem to have become progressively worse as this story is told. I get the idea that she is not very sick.

Appendicitis, the commonest lesion statistically, as an explanation for this picture has a few things wrong as a diagnosis in this case. She did not begin with pain in the epigastrium or generalized pain. It was localized in the right lower quadrant from the beginning. She was awakened with sudden pain instead of having prodromal symptoms of something getting worse and worse. This story of vomiting twice a week before onset and being perfectly well in between is certainly not at all characteristic of appendicitis and why she vomited the week before, I have not the faintest idea. There were previous attacks of something 5 or 6 years ago which may have been attacks of appendicitis but nothing is said to give us any help on that. Her temperature and white count are compatible with acute appendicitis. The rectal tenderness is compatible with pelvic appendicitis and the absence of vaginal tenderness is compatible. So that although we have to be prepared to be wrong in the diagnosis, I think there is no question as to what this treatment should be. It is obviously something that requires operation, and it is probably acute appendicitis in the pelvis.

Clinical Diagnosis

Subsiding appendicitis.

Dr. Horatio Rogers's Diagnosis

Subsiding pelvic appendicitis.

Pathologic Diagnoses

Follicular cyst of the ovary with rupture.
Healing appendicitis.
Parovarian cyst.

Pathologic Discussion

Dr. Mallory: The house officer who operated on this case has finished his service, so I will have to describe his findings from the records. As he approached the region of the pelvis he found large amounts of blood on both sides, some old and clotted, some fairly fresh. The appendix was retrocecal and entirely out of the picture. The left ovary contained a fairly large ruptured follicular cyst. There was nothing present in the right ovary and I think there is no question that a ruptured follicular cyst with hemorrhage was the cause of her symptoms. It is strange, first, that it should have occurred at this very early period of the cycle, and, secondly, that since the cyst was in the left ovary that, from beginning to end, her symptoms were on the right.

Dr. Ralph Adams: I saw that operation and it explains the right lower quadrant symptoms if one knows that the left ovary lay behind and to the right of the uterus.

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A CHRISTMAS MESSAGE

As the swift seasons roll, we arrive, more or less automatically, at that particular stage of the annual cycle when we begin to wonder what Saint Nicholas has in the bag for us. We in our own profession and, we are told, those outside of it as well have become somewhat accustomed in the last few years to receiving prize packages, not always of pleasant content and not always coming at the merry Yuletide.

If we are philosophically minded, those jabs in our various tender spots have served after a sort to immunize us against adverse social or economic substances; if we are of a type destined to make us the sports of Fate we have become sensitized, which is a different and less pleasant matter. In this sadder case succeeding injections serve only to arouse reactions undesirable in their nature and still more irritating in their effects. Who, after all, wants to become allergic to environmental conditions over which he has no control, or exhibit only reactions of sensitivity to circumstances which he is capable of improving?

Such thoughts, however, savor of a paternal if not, indeed, a didactic philosophy and are undoubtedly a little too deep for your mere editorial writer to toy with. As Confucius might have said, he who cannot swim must wade, and waders should stick to shallow water unless they want to get wet all over, and that bottoms up. Moreover, the *Journal*, despite its hundred odd years of observation of matters medical (and animal and vegetable and mineral, as those who have lived through the Gilbertian era might be tempted to put it) does not yet feel itself of an age to attempt either pulpit preachment or peripatetic pedagogy.

What we meant to say was that there are certain gifts in the bag of Saint Nicholas for all of us if we will reach in and pull them out, and rather than three wise men bringing gifts there will be many receiving them. There is the gift of silence when mere words are superfluous—an inexhaustible gift for all who would make use of it; there is the gift of sympathy and kindness toward those who are dependent upon us for good cheer in times of trouble; there is the gift of fairmindedness and ethical consideration toward our colleagues; there is the gift of holding a respected place in a profession respected above all others, and increasing the respect in which it is held among the multitudes of all nations.

With such gifts at our disposal we cannot fail to enjoy the Merry Christmas and the Happy New Year which the *Journal* wishes to its goodly company of readers.

ANNUAL REGISTRATION OF PHYSICIANS

THE Board of Registration in Medicine has again advocated the re-registration of physicians and has introduced a Bill providing that such registration be required annually. The Bill differs in two respects from that introduced last year, namely, in making the fee one dollar instead of two, and in characterizing and limiting the information about the physician which the Board may require and publish. The purpose of the information is to identify the physician, but not in every respect. It is merely in his professional capacity, so that his registration number, his professional address, his medical education, his field of practice (specialty) and special method of treatment employed, if any (Homeopathy, Osteopathy, Eclecticism or other system), are all pertinent and may be helpful to the lay person seeking information about a physician to the end that he may be well informed when he makes his choice. Whether he chooses more wisely is another question.

Why is such a bill advocated? As was stated

last year, the Board of Registration in Medicine acting under the statute registers physicians and preserves its register of names which is a matter of public record. No provision is made under the existing statute for revising the register from time to time. The only information concerning a registered physician that comes to the Board (after the Town Clerk notifies the Board as to the local registration) is on complaint or on enquiry, as from an insurance company or credit agency or some other interested person. If the physician dies, only by chance does the Board learn of his cessation of practice.

The purpose of the Bill, then, is first to provide an accurate and up-to-date register of duly licensed physicians practicing in the Commonwealth; and, secondly, to make this register easily available by publishing copies for distribution throughout the State.

Is such a list of practical value? Certainly the publication of the list is no guarantee that it will be used, but the results in states where such a list is widely distributed indicate that people really do want to know more about the qualifications of their physicians, and if they know that a person is not registered by the state, they are less likely to seek his professional assistance. According to the experience of other states, many of the unlicensed practitioners of medicine voluntarily leave promptly when such a bill, as is advocated for Massachusetts, becomes law.

Are there then no objections to the Bill? There are indeed objectors and they give several reasons. The first objection is on the ground of unconstitutionality, but although such laws have been on the statute books of nearly twenty states, in some states for a number of years, the question has never been brought into court for decision, and legal opinion is against objection on the ground of unconstitutionality.

The second objection is on the part of some members of the medical profession who, as they express it, resent a tax on reputable physicians designed to employ the police in doing what they ought to do anyhow. An additional tax on physicians is not to be advocated with the glib assurance that the doctor is always ready to help any good cause. But these objectors fail to distinguish between the "police" and the "police power of the state". The whole work of the Board of Registration in Medicine (except in so far as it may be incidentally educational) is under the police power of the State, while it is clearly not under the police. The main purpose of such exercise of the police power of the State as would be involved in carrying out the provisions of the bill would be educational, in disseminating correct information throughout the State concerning registered physicians. Incidentally it would show up by contrast persons not licensed, who would be the more easily compelled to become registered if qualified, or to leave the State if they desired to continue in their practice without registration, or to desist from practice if they continued to reside in Massachusetts.

The economic side is not too unimportant to be mentioned. The cost of registration might be seven thousand dollars a year for the whole medical profession. It has been estimated that there are a thousand persons in Massachusetts practicing medicine in some form without being licensed. The enlightenment which would come from having an authoritative list of licensed physicians (a legitimate form of advertising) easily accessible in all parts of the State, might reasonably be expected to deflect an amount of money at least equal to the cost of registration into the depleted moneybags of the medical profession.

The text of the bill is appended:

AN ACT providing for the Annual Registration of Physicians and the Annual Publication of the List of Physicians duly registered.

Section 1. Chapter one hundred and twelve of the General Laws, as appearing in the Tercentenary Edition thereof, is hereby amended by inserting after section four the two following new sections:—

Section 4A. Every person registered by the board as a qualified physician, who is engaged in the practice of medicine within the commonwealth, shall annually in December renew his registration for the ensuing calendar year by payment of one dollar to the board and recording with the board his name, his registration number, his professional address, and such other identifying information concerning his medical education as the board may require, together with the field of his practice, including the special system of treatment employed, if any, on blanks furnished by the board at the request of the physician and signed by him under the penalties of perjury; and thereupon the board shall issue to him a certificate showing that he is entitled to continue in the practice of medicine for the period covered by said renewal. Whoever, being duly registered under section two or corresponding sections of earlier laws, practices or attempts to practice medicine without complying with the requirements of this section, shall be punished by a fine of not less than five nor more than one hundred dollars.

Section 4B. On the first day of March of each year, the Board shall publish a list of the physicians who, in compliance with the provisions of section two or section four A, as the case may be, are authorized

by the board to engage in the practice of medicine in the commonwealth during the current calendar year, giving the name of each registrant, his registration number, his professional address, and such other identifying information as is specified in section four A, and shall send to each registrant a copy thereof.

THE INDISPENSABLE VITAMIN

PARALLEL with a deplorable amount of commercial ballyhoo concerning the vitamins, much serious and valuable investigative work continues to go forward. Vitamin D continues as one of the favorite children of the advertising brain trust, although actual knowledge as to its exact effects and dosage is still so uncertain that authorities at the University of California Medical School and the San Francisco Health Department consider inadvisable "any attempt to increase the sale of a product, especially milk, by the addition of a substance whose value to the public health is still a controversial problem".

The manufacturers of proprietary remedies have some time since become vitamins A and B conscious; their contentions based not so much on the ill-effects of a definite deficiency in these substances, as on the positive benefits to be derived from very large doses of them; vitamin A, for instance, increases the tissue's resistance to infection, and vitamin B, according to optimistic reports, stimulates the appetite to hitherto unknown proportions. Vitamin E, the fertility vitamin, the latest to be brought before the public, not only prevents abortion but erases adolescent acne as if by magic.

Vitamin C, the oldest known and possibly the most important of the accessory substances has so far received its ballyhoo mainly from the producers of natural foods. The human race, as the report of President Hoover's White House Conference stated, lives from hand to mouth so far as its supply of ascorbic acid is concerned, for it depends for this upon its annual crops of fresh fruits and vegetables. As a consequence the comparatively recent isolation of this acid and its synthesization from glucose may have more far-reaching effects on the welfare of the race than the work which has been done on all the other vitamins put together.

The chief obstacle in the way of preserving vitamin C for future needs has lain in its susceptibility to destruction by oxidation. Paul F. Sharp, writing in *Science*, tells us that this susceptibility to oxidation depends partly on the fact that plant tissues containing ascorbic acid apparently contain also an ascorbic acid oxidizing enzyme which is liberated when the cells are crushed. The large amount of ascorbic acid present in cabbage, for instance, is completely oxidized within five minutes after the previously frozen raw cabbage cells are disintegrated. Ruminants, consequently, despite the large amounts of ascorbic acid ingested, must either synthesize the acid for their uses, or reverse the oxidation process. It is known that such animals as rats, whose diets rarely contain ascorbic acid, are able to synthesize it from glucose, the basic product from which commercial ascorbic acid is now manufactured.

Sharp points out that the ascorbic acid content of fresh cow's milk varies little through the seasons. Milk also, however, contains an oxidizing enzyme which eventually destroys the acid. Pasteurization, instead of accelerating oxidation as has been usually believed, may actually inhibit it, since under proper conditions more of the enzyme than of the ascorbic acid is destroyed by heat. The presence of copper, however, accelerates oxidation unless the temperature reached is high enough practically to destroy the enzyme completely. These investigations emphasize the necessity, if ascorbic acid is to be preserved in milk, of banishing copper from pasteurizing machines and holding vats and it is doubtful if even under the best conditions milk can be relied upon to contain a protective amount.

Fresh milk has been found to contain 20.1 mg. ascorbic acid per liter. After being held for three days at 2° C. this figure is found to have dropped to 11.3 mg.; if the milk has been pasteurized for 30 minutes at a temperature between 62 and 63° C. the figure is 11.0 mg.; if copper is present the ascorbic acid content will have dropped to 1.7 mg.

The antiscorbutic needs of the human infant are not definitely established, but it is probable that at least 20 mg. a day are necessary to maintain a margin of safety.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MINOT, GEORGE R. A.B., M.D. Harvard University Medical School 1912. S.D., F.R.C.P. (Edin.) Professor of Medicine, Harvard University Medical School. Director, Thorndike Memorial Laboratory, and Visiting Physician, Boston City Hospital. Member, Board of Consultation, Massachusetts General Hospital. Consulting Physician, Peter Bent Brigham and Beth Israel Hospitals, Boston. His subject is "Harvard and Nutrition." Page 1147. Address: Thorndike Laboratory, Boston City Hospital, Boston, Mass.

GAMBLE, JAMES L. A.B., S.M., M.D. Harvard University Medical School 1910. Professor of Pediatrics, Harvard University Medical School. Visiting Physician, Children's Hospital and Infants' Hospital, Boston. His subject is "Extracellular Fluid and Its Maintenance." Page 1150. Address: 300 Longwood Avenue, Boston, Mass.

JONES, CHESTER M. B.A., M.D. Harvard University Medical School 1919. Assistant Professor of Medicine, Harvard University Medical School. Physician, Massachusetts General Hospital. Consultant in Medicine, Newton Hospital. His subject is "Protein Deficiency." Page 1152. Address: Massachusetts General Hospital, Boston, Mass.

HEATH, CLARK W. A.B., M.D. Harvard University Medical School 1926. Instructor in Medicine and Assistant Medical Adviser, Harvard University Medical School. Assistant Physician, Thorndike Memorial Laboratory, Boston City Hospital. His subject is "Mechanism of Hemoglobin Deficiency." Page 1155. Address: 319 Longwood Avenue, Boston, Mass.

CASTLE, WILLIAM B. M.D. Harvard University Medical School 1921. Associate Professor of Medicine, Harvard University Medical School. His subject is "The Relationship of Defective Nutrition to Changes in the Gastrointestinal Tract." Page 1158. Address: Boston City Hospital, Boston, Mass.

WOLBACH, S. BURT. M.D. Harvard University Medical School 1903. Shattuck Professor of Pathological Anatomy, Harvard University Medical School. Pathologist, Peter Bent Brigham, Children's, Infants', and Boston Lying-in Hospitals. His subject is "Vitamin C and the Formation of Intercellular Material." Page 1158. Address: Harvard University Medical School, 240 Longwood Avenue, Boston, Mass.

BLACKFAN, KENNETH D. M.D. Albany Medical College, Union University, 1905. Thomas Morgan Rotch Professor of Pediatrics, Harvard University Medical School. Chief of the Medical Service, Children's Hospital. Medical Director, Infants' Hospital, Boston. His subject is "Progress in the Early Recognition of Vitamin Deficiency States." Page 1159. Address: 300 Longwood Avenue, Boston, Mass.

HOWE, PERCY R. A.B., D.D.S. Philadelphia Dental College 1890, D.Sc. Alexander Forsyth Professor of Dental Science, Harvard Dental School. Instructor in Pathology, Harvard University Medical School. Director, Forsyth Dental Infirmary for Children. His subject is "Oral Pathology in Relation to Avitaminosis." Page 1163. Address: 49 Cedar Road, Belmont, Mass.

STRAUSS, MAURICE B. A.B., M.D. Johns Hopkins University School of Medicine 1928. Instructor in Medicine, Harvard University Medical School. Research Fellow, Thorndike Memorial Laboratory, and Assistant in Medicine, Boston City Hospital. His subject is "Nerve Disorders Arising from Defective Nutrition." Page 1164. Address: 270 Commonwealth Avenue, Boston, Mass.

JOSLIN, ELLIOTT P. B.A., M.A., M.D. Harvard University Medical School 1895. Clinical Professor of Medicine, Harvard University Medical School. Medical Director, George F. Baker Clinic, New England Deaconess Hospital. Consulting Physician, Boston City Hospital. His subject is "Protamine Insulin and Its Advantages." Page 1166. Address: 81 Bay State Road, Boston, Mass.

PEMBERTON, FRANK A. S.B., M.D. Harvard University Medical School 1909. F.A.C.S. Clinical Professor of Gynecology, Harvard University Medical School. Surgeon-in-Chief, Free Hospital for Women, Brookline, Mass. Address: 198 Commonwealth Avenue, Boston, Mass. Associated with him are

SMITH, GEORGE VAN S. A.B., M.D. Harvard University Medical School 1926. Visiting Surgeon, Pathologist and Director of Research, Free Hospital for Women and Fearing Research Laboratory, Brookline, Mass. Research Fellow in Gynecology, Harvard University Medical School. Address: Free Hospital for Women, Brookline, Mass. And

GRAVES, SIDNEY C. A.B., M.D. Harvard University Medical School 1929. Assistant Visiting Surgeon, Free Hospital for Women, Brookline, Mass. Address: 198 Commonwealth Avenue, Boston, Mass. Their subject is "Urinary Fistulas Opening Into the Vagina." Page 1170.

WITHINGTON, PAUL R. M.D. Harvard University Medical School 1916. Visiting Physician, Milton Hospital. Clinical Assistant Physician, Children's Hospital. President, Massachusetts Public Health Association. His subject is "Moulded Plaster Right Angle Elbow Splints." Page 1174. Address: 350 Randolph Avenue, Milton, Mass.

The Massachusetts Medical Society

NEW FELLOWS ADMITTED TO THE DISTRICT SOCIETIES IN NOVEMBER, 1936

Berkshire

Beers, Daniel Nichols, 74 North Street, Pittsfield.

Bristol North

Seguin, Wilfrid Adrien, State Hospital, Taunton.

Bristol South

Buck, Roger, 262 Cherry Street, Fall River.
LaSalle, Arthur, 1659 County Street, Somerset.
Lawson, Henry Carnie, 124 Franklin Street, Fall River.
Merchant, Raymond Francis, Main Street, Vineyard Haven.
Nye, Lucius Seymour, 15 Sawyer Street, Wareham.

Essex South

Fraser, William Gardiner, 41 Atlantic Street, Lynn.
Hinchey, Paul Richard, 355 Essex Street, Salem.
Mutty, Lawrence Theodore, 54 Union Street, Manchester.
Piotrowski, Walter Louis, 9 Washington Street, Peabody.
Winer, Irving Morton, 4 Washington Street, Peabody.

Franklin

Strong, Paul Theodore, Mount Hermon.
Thomas, Warren Dustin, Montague.
Thorn, Edwin Charles, Greenfield.

Hampden

Ashton, Paul Millner, 11 High Street, Springfield.
Canter, Bernard, 663 State Street, Springfield.
Daniels, Marie Hurwicz, 239 Madison Avenue West, Holyoke.
Duffy, Frank King, Mercy Hospital, Springfield.
Feldman, Noah Norman, 176 Chestnut Street, Holyoke.
Mahoney, Frank Aloysius, Jr., Springfield Hospital, Springfield.
Popkin, Norman, 425 Trafton Road, Springfield.
Ross, George Lewis, 139 Chestnut Street, Holyoke.
Schiff, Joseph, 10 Chestnut Street, Springfield.
Simpson, Howard Nellson, Springfield Hospital, Springfield.
Strachan, Harry Leitch, Jr., Springfield Hospital, Springfield.
Tillman, Henry Bernard, 40 Washington Street, Springfield.
Weisman, Jacob Irving, 116 Fort Pleasant Avenue, Springfield.

Hampshire

Burrier, Walter Painter, United States Veterans Facility No. 95, Northampton.
Donais, Henry Edward, 49½ Union Street, Easthampton.

Middlesex East

Devine, James Anthony, 14 Winn Street, Woburn.
Larsen, Carl John, New England Sanitarium, Stoneham.
McNamara, Francis Joseph, 478 Lebanon Street, Melrose.
Turtle, William John, 5 Oneida Circle, Winchester.

Middlesex South

Abbott, John Adams, Sudbury Road, Concord.
Arne, James Alexander, 352 Main Street, Medford.
Biller, Albert Livingston, 3 Otis Street, Watertown.
Brusch, Charles Armao, 843 Massachusetts Avenue, Cambridge.
Corkery, James Richard, 116 Jefferson Avenue, Everett.
Farrington, Ralph Curtis, 50 Lexington Street, Framingham.
Franseen, Clifford Carlton, 30 Kinross Road, Brighton.
Garcelon, Gerald Goodwin, 51 Pickwick Road, West Newton.
Glendy, Margaret Moriarty, 219 Commonwealth Avenue, Newton.
Glendy, Robert Earle, 219 Commonwealth Avenue, Newton.
Green, Leo Arthur, Cambridge City Hospital, Cambridge.
Guidotti, Hugo George, 14 Central Street, Somerville.
Holoff, James, 96 Forest Street, Medford.
Hubbard, John Perry, 9 Wyman Road, Cambridge.
Huber, Edward Godfrey, 45 Homewood Road, Waban.
Kavalgian, Aram, 553 Mt. Auburn Street, Watertown.
Kennard, Harrison Eisenbrey, 246 Dudley Road, Newton.
Larson, Carl Gustav, 119 High Street, Medford.
Rudy, Harold Aaron, Cambridge City Hospital, Cambridge.
Stewart, John Dunham, 410 Memorial Drive, Cambridge.
Sullivan, Frank Joseph, 9 Cowperthwaite Street, Cambridge.

Norfolk

Brenner, Harry Harold, 973 Main Street, Walpole.
Chamberlin, Donald Tillinghast, 368 Longwood Avenue, Roxbury.
Devlin, William James, 42 Arborway, Jamaica Plain.
Dunphy, John Joseph, Jr., 10 Dix Street, Dorchester.
Fisher, Alexander Newman, 25 Esmond Street, Dorchester.
Flanagan, Norris Butler, 47 Walk Hill Street, Forest Hills.
Fleming, Robert Edward, 12 Lowell Road, Brookline.
Gaston, Eugene Alexander, 16 Cocasset Street, Foxborough.
Massell, Benedict Frank, 25 Binney Street, Roxbury.
Michaels, Joseph Jules, 214 Riverway, Roxbury.
Morse, Fred Winslow, Jr., 11 Fells Road, Wellesley.
Mullane, Daniel Joseph, 776 Centre Street, Jamaica Plain.
Musche, Frank Wilbur, 30 Milton Street, Dedham.
Schwab, Robert Sidney, 74 Fenwood Road, Roxbury.
Semrad, Elvin Vavrinec, 74 Fenwood Road, Roxbury.
Stone, Edward Stephen, 27 Stearns Road, Brookline.
Twadelle, Frank Joseph, 192 Washington Street, Wellesley.
Walter, Carl Waldemar, 721 Huntington Avenue, Roxbury.
Whelan, Joseph David, 67 Maple Street, Needham.

Norfolk South

Catinella, Paul Joseph, Quincy City Hospital, Quincy.
Cohen, Archibald Clinton, 808 Broad Street, East Weymouth.

Stein, Max Donald, Quincy City Hospital, Quincy.
Whitehurst, Clinton Howard, 839 Nantasket Avenue, Allerton.

Plymouth

Farren, Edward Bain, 231 Main Street, Brockton.
Lydon, Mark Thomas, 352 North Avenue, North Abington.

Suffolk

Beecher, Henry Knowles, 34 Cedar Lane Way, Boston.
Diez, Mary Luise, 22 Lynde Street, Boston.
Doyle, Richard Joseph, Boston City Hospital, Boston.
Easton, Mahlon Tracy, 209 Newbury Street, Boston.
Garrey, Walter Easton, 279 Clarendon Street, Boston.
Gobert, Solomon, 141 Meridian Street, East Boston.
Harberson, James Coulter, 330 Dartmouth Street, Boston.
Jeghers, Harold Joseph, Boston City Hospital, Boston.
Kannan, Michael Joseph, Boston City Hospital, Boston.
Karp, David, Boston Floating Hospital, Boston.
Leech, John Verner, 65 Bay State Road, Boston.
MacEwen, Herbert Bruce, 270 Commonwealth Avenue, Boston.
Matarese, Antonio Andrew, Boston City Hospital, Boston.
McKay, Florence Lucinda, 133 Mt. Vernon Street, Boston.
Morrissey, Arthur Michael, 818 Harrison Avenue, Boston.
O'Brien, Joseph Vincent, Boston City Hospital, Boston.
Schwartz, Louis Nathaniel, Boston City Hospital, Boston.
Selesnick, Sydney, 818 Harrison Avenue, Boston.
Spink, Wesley William, Boston City Hospital, Boston.
Sullivan, William Joseph, Boston City Hospital, Boston.
Weller, Alfred, Boston Floating Hospital, Boston.

Worcester

Contino, Charles Anthony, Worcester. (Temporary address: 90 Main Street, Leominster.)
Cutler, Isadore Louis, Rutland State Sanatorium, Rutland.
Dunlop, George Rodgers, 27 Elm Street, Worcester.
Dunn, Raymond Anthony, 504 Pleasant Street, Worcester.
Ferrucci, Joseph, 21 Court Street, Milford.
Folsom, Hugh Francis, Southborough.
Massell, Theodore Benedict, 30A Boynton Street, Worcester.
Pelecovich, Marie, 574 Main Street, Shrewsbury.
Seelye, Edwin Barrows, 390 Main Street, Worcester.
Showstack, Nathaniel, Grafton State Hospital, North Grafton.
Zehner, Minerva Caroline, 36 Pleasant Street, Worcester.

Worcester North

Lilly, Edward, Davis Street, Shirley.
Perkins, Beatrice Louise, 20 Parker Street, Gardner.
Savignac, Eugene Martin, 66 Parker Street, Gardner.

MISCELLANY

HEALTH INSURANCE STUDY IS INITIATED BY SECURITY BOARD

The Social Security Board has initiated a study looking to possible proposal of a major addition to the social security system in the shape of health insurance, it was disclosed recently.

As large a Federal project for social welfare as either unemployment insurance or the old-age benefit insurance system, health insurance would provide both medical services and cash payments in partial compensation for wage losses due to illness. Should legislation for this purpose be sponsored, it would probably be of the same universal type as the old-age benefits. The coverage would extend to most of the working population of the nation and taxes would be required to finance it.

EARLY PROPOSAL DELAYED

An official proposal considered two years ago, but postponed, contemplated establishment by the Federal government of minimum standards for health insurance practice and provision of grants or other incentives to states undertaking the development of systems meeting the Federal standards. The fact is cited that nearly every large industrial country of the world has applied the principle of insurance to the risks of illness.

The new study is in line with recommendations made by Harry Hopkins, WPA Administrator, in a recent speech to the United States Conference of Mayors, and the report of the executive council of the American Federation of Labor.

A spokesman for the Social Security Board said that the board had drawn no bill on the subject and had made no commitment as to sponsoring the new line of insurance. Invoking the authority of Section 702 of the social security act, however, the board has directed that a research study of the subject be made and experts are starting work in this connection, it was stated. In view of the promise of President Roosevelt to avoid new taxes at the coming session of Congress, it is possible that the Social Security Board may temporarily withhold pressing the health insurance proposal even if it should decide to recommend favorable action on the basis of the projected studies.

COST ESTIMATE IS HIGH

Comprehensive health insurance has been estimated as likely to cost the equivalent of more than 5 per cent of pay rolls. The President's committee on economic security in January, 1935, prepared a tentative plan, but recommended further studies. It found that the cost of wage-loss payments would be 1 to 1¼ per cent of pay roll. It estimated that families

with up to $3,000 earnings a year are spending 4½ per cent now of their income for medical care. Under existing social security legislation the unemployment insurance tax will run to 3 per cent of pay rolls, payable by the employer, and the old age benefits will cost 6 per cent, shared equally by employers and employees. State and Federal money, however, is already being raised and spent for a certain amount of medical care which would be covered in the service offered by healtn insurance. It is held improbable that any recommendation would contemplate raising all the cost of health insurance from pay roll taxes.

The Security Board's study will be under the general direction of Walton Hale Hamilton, economist head of the research division and former member of the National Industrial Recovery Board.

Existing systems of unemployment compensation and old-age benefits are generally believed in Security Board circles as sure to bring health insurance to the fore.

Unlike unemployment compensation and old-age benefits, health insurance requires the building up of no large reserve, officials say. The plan can be placed on a pay-as-you-go basis, each year's cost being met out of the year's receipts. Also, unlike the other two insurance plans already established, the insured worker under health insurance gets benefits from the start. He does not have to wait until the next depression or until he reaches old age. Accordingly, it is believed there is less difficulty in the collection of the taxes or contributions than in these other forms of insurance.—Excerpts from an article in the N. Y. *Herald-Tribune*, Nov. 23, 1936.

ACTIVITIES OF THE AMERICAN SOCIAL HYGIENE ASSOCIATION

Plans for the first National Social Hygiene Day, to be held February 3, 1937, are announced by the American Social Hygiene Association, of 50 West Fiftieth Street, New York City. On this day, state and community voluntary organizations interested in the control of syphilis and gonorrhea and other social hygiene problems, with the advice and approval of health authorities and the medical and allied professions, are planning to hold meetings all over the United States.

In New York City, the American Social Hygiene Association will hold its annual meeting on February 3. Also the Social Hygiene Council of Greater New York will hold its Fifth Annual Regional Conference at the Hotel Pennsylvania on the same day. It is expected that public leaders, including Surgeon General Parran, President Ray Lyman Wilbur of Stanford University, President of the American Social Hygiene Association and former Secretary of the Interior, will speak to these meetings. National agencies and many of their state and community organizations which include social hygiene activities in their yearly programs are planning to participate. It is probable that a nation-wide radio hook-up will provide addresses of great importance from high government officials and civic leaders in different parts of the country as a climax to the activities of the First National Social Hygiene Day.

There has been definite progress all along the line during the past year in public understanding and support of the campaign against syphilis. Newspapers and magazines are opening their columns to public discussion of this health menace to a greater extent than ever before. Certain important groups, such as the General Federation of Women's Clubs and the National Council of Women, are adopting the fight against syphilis as among their next major efforts in promoting community health. (The women's groups are particularly interested in the elimination of prenatal or congenital syphilis, which, acquired by a child before birth from an infected mother, is responsible for a large share of stillbirths, miscarriages and defective children, and which is entirely preventable by proper treatment.)

Service luncheon clubs, such as Rotary, Kiwanis, and Lions, have recently been undertaking social hygiene programs. Business leaders are studying the cost to industry, from lost time, lowered efficiency and hospitalization due to syphilis. The large insurance companies are concerned over the unnecessary claims for death and disability due to syphilis. Civil clubs, forums and town meetings are discussing the diagnosis and treatment of syphilis as a national plague.

It is believed that the direction of united nation-wide attention to this subject in the way that is proposed will help professional and lay community leaders to capitalize and increase this new interest, and consolidate for further advance toward meeting General Parran's challenge to "stamp out syphilis".

VOLUNTARY HOSPITAL INSURANCE

A gift of $100,000 to the American Hospital Association for the study and development of voluntary hospital insurance was announced by Edwin R. Embree, president of the Julius Rosenwald Fund, at the annual meeting of the Fund recently held in Chicago. This plan, known as group hospitalization, enables persons of moderate means to secure hospital care by payments of from $6 to $12 per year without recourse to charity.

The program of the American Hospital Association will be carried forward through a special Committee on Hospital Service of which C. Rufus Rorem of Chicago becomes executive director. The chairman of the Committee is Dr. Basil C. MacLean of Rochester, New York, and other members are Dr. R. C. Buerki, Madison, Wisconsin; Dr. S. S. Goldwater, New York City; Msgr. Maurice F. Griffin, Cleveland, Ohio, and Dr. Claude W. Munger, president of the American Hospital Association.

The work of the Committee on Hospital Service includes two phases: first, advice and consultation to existing plans and those being formed concerning

actuarial data, benefits, methods of organization, public relations, annual subscription rates; secondly, relations of hospital service plans to the medical profession, public welfare activities, state departments of insurance, private insurance companies, hospital administration, and hospital accounting. This program is a continuation of the activities of the American Hospital Association since 1933.

Dr. Rorem, who is a certified public accountant, was formerly associate professor at the University of Chicago and is the author of a university text in accounting as well as several volumes dealing with the economic and financial aspects of hospital and medical care. Since 1931 he has been associate director for medical services of the Julius Rosenwald Fund and since 1933 has been consultant in group hospitalization to the American Hospital Association.

Enrollment in group hospitalization plans is now approaching one-half million employed subscribers and dependents with more than 150,000 participating in the three-cents-a-day plan for hospital care in New York City. Plans which have enrolled more than 25,000 employed persons are those in Rochester, New York; Cleveland, Ohio; Washington, D. C.; Minneapolis and St. Paul, Minnesota, and Dallas, Texas. Other plans with 5,000 or more subscribers and dependents are those in New Orleans, Syracuse, St. Louis, San Antonio, Houston, Memphis, Sacramento, Newark, Charleston and Bluefield, West Virginia; Kingsport, Tennessee, and a state-wide plan for North Carolina. Nonprofit city-wide hospital service plans have been established or are being organized at the present time in Chicago, Buffalo, Albany, Louisville, New Haven and Boston.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1935 AND SEVEN YEAR AVERAGE

MONTH ENDING DECEMBER 5, 1936

Diseases	1936					1935			
	Week ending Nov. 14	Week ending Nov. 21	Week ending Nov. 28	Week ending Dec. 5	Average cases reported for week corresponding to Dec. 5 for past seven years	Week ending Nov. 16	Week ending Nov. 23	Week ending Nov. 30	Week ending Dec. 7
Chickenpox	75	130	102	161	146	96	121	89	149
Conjunctivitis Infectious	3	—	—	2	—	—	—	—	—
Diphtheria	1	6	1	4	14	3	1	4	9
Dysentery Bacillary	2	1	—	1	—	1	—	—	—
Encephalitis Epidemic	—	—	—	1	—	1	—	—	—
German Measles	3	3	2	7	6	9	13	19	4
Influenza	5	4	1	4	5	3	2	18	9
Measles	22	75	43	75	75	52	55	29	110
Meningococcus Meningitis	—	—	—	1	—	1	1	2	—
Mumps	45	107	54	51	50	29	63	45	65
Paratyphoid Fever	3	1	1	1	—	—	2	1	—
Pneumonia (Broncho)	42	30	26	31	30	19	9	12	24
Pneumonia (Lobar)	37	33	18	29	32	17	18	13	34
Poliomyelitis	—	2	—	—	—	3	6	5	2
Scarlet Fever	38	35	32	44	60	27	54	32	33
Smallpox	—	—	—	—	6	—	—	—	—
Streptococcus Sore Throat	1	1	2	4	1	2	1	—	1
Tetanus	—	—	1	—	—	—	—	—	—
Trachoma	—	1	—	—	—	—	—	—	—
Trichinosis	—	—	—	1	—	2	—	—	2
Tuberculosis (Pul.)	31	19	19	13	24	23	22	13	26
Tuberculosis (O. F.)	—	2	—	—	2	2	1	1	3
Typhoid Fever	—	3	2	1	3	2	1	—	1
Undulant Fever	1	3	3	3	—	3	2	4	3
Whooping Cough	124	151	72	119	64	82	110	75	71
Gonorrhea	23	20	26	33	50	27	34	26	22
Syphilis	38	31	49	65	58	26	42	37	49

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

INCREASE IN CANCER DEATHS IS APPARENT, NOT REAL

Those who have been worrying over the menace of an increasing cancer death rate can ease their fears and take heart from the optimistic note on cancer struck by the latest figures of the Metropolitan Life Insurance Company.

The increase in the cancer death rate during recent years is more apparent than real. No more people are dying of cancer now than 25 years ago, but more cancer deaths are being recorded because of better diagnosis. In certain groups, notably white women between the ages of 35 and 54, the death rate has declined significantly in the past 25 years. Only over the age of 65 has the cancer death rate for white women shown an upward trend.

These are among the findings of a survey of cancer deaths among the company's industrial policyholders during the past quarter century.

Even among white males the cancer death rate is not quite a third higher than that of 25 years ago. This increase, it is believed, is due to improved diagnosis, more cases being recognized now as a result of improvements in modern medicine during the past quarter century. These improvements in diagnosis have caused an apparently greater increase in the cancer death rate for men than for women in the 25 years, because in men cancer occurs more often in internal organs where it was not easily accessible for diagnosis.

Bearing out this point which argues that the increase in cancer deaths is more apparent than real is the fact that the recorded death rates from cancer in accessible places have declined while those from inaccessible cancers have increased.

Other hopeful developments in the cancer situation in the United States are the growth of facilities both public and private for treating cancer and the improvement, as a result of special training, of the physician's ability to diagnose and treat cancer effectively.

Also encouraging is the fact that between 1932 and 1935 the American College of Surgeons collected data on 25,000 patients living five or more years after treatment for cancer without recurring signs of the disease.

The true death rate from cancer may not have increased, but cancer still remains a major public health problem, the life insurance company's statistician, Dr. Louis I. Dublin, points out. Cancer ranks second only to heart disease as a cause of death, and still takes 135,000 lives each year.—*Science News Letter*, November 14, 1936.

LOCAL HEALTH OFFICERS CAN FIGHT MENTAL DISEASE

People in all ages and in all walks of life may need help in protecting themselves from mental ill health, Dr. B. Liber, New York City psychiatrist, indicated. He pointed out that preventing mental disease is as much a problem for public health agencies as preventing smallpox or other physical disease.

Cooperation of trade unions and of employers should be gained by health officers in order to protect the mental health of workers, Dr. Liber said. Employers should learn that it is to their own advantage that the workers be clear-minded, alert and calm in order to prevent accidents and to produce more and better quality work.

"Child upbringing, sexual problems in youth, marital problems and industrial difficulties constitute the worst causes of mental maladjustment," Dr. Liber said.

Other methods by which health departments can protect mental health as suggested by Dr. Liber are the following:

1. Preventive mental clinics for transition cases of adults.
2. Mental hygienists in all elementary public schools.
3. At least one mental examination of all pupils.
4. Behavior problems solved in cooperation with parents.
5. More attention to mental hygiene in schools for teachers.
6. More and better Child Guidance Clinics.
7. Easy courses in child upbringing for parents and future parents, preferably in public school buildings.
8. Marital consultation bureaus, not compulsory, but friendly, sympathetic and confidential for young men and women before marriage. Discouraging unions between families where insanity or feeblemindedness prevails. Discovering gonorrhea, syphilis, tuberculosis in candidates for marriage.
9. Classes for adolescents in sex education. Dispelling fears and anxieties due to ignorance and leading to mental maladjustment.—*Science News Letter*, November 14, 1936.

THIRTY-FIVE PER CENT SUCCESS IN USE OF SPRAY FOR POLIOMYELITIS

About one-third of those persons who used the alum picric acid nasal spray as a preventive of infantile paralysis during the epidemic in Southern states last summer were protected against the disease by this spray.

This appears from the first report of results with the spray. The report was given by Dr. Charles Armstrong, U. S. Public Health Service officer, who developed the spray, at the meeting of the southern branch of the American Public Health Association.

While these results are somewhat disappointing to Dr. Armstrong and his associates, investigations are now under way which it is believed will give a more effective method of prevention in time for use next summer.

The spray was used on a large scale on children and young adults during the outbreak in the South last summer. Since September, Dr. Armstrong has been gathering reports on its use. The fact that it was used by laymen as well as physicians com-

plicated the situation considerably, because the layman in many cases did not use the spray so effectively and thoroughly as a physician would have. The results show, among other things, that the method is not suitable for use except by physicians, Dr. Armstrong commented.

A house-to-house survey was made of 20 representative districts in Birmingham and 7 districts in surrounding Jefferson County. In this group, 5,010 persons out of a total of 8,093 used the spray at least once. (The directions were to use it every other day for a week and then once a week for the duration of the epidemic.) If the same rates prevailed in the entire area of Birmingham city and Jefferson County, 270,000 persons were sprayed and 160,000 were not, it is calculated.

In the sprayed group, 7 cases of infantile paralysis developed. In the unsprayed group, 8 cases developed. Calculating the number among the sprayed group who might have been expected to get the disease on the basis of the percentage among the unsprayed who developed it, Dr. Armstrong got a ratio of 16 to 21.7. This indicates that about one-third, or 35 per cent, were protected by the spray.—*Science News Letter*, November 28, 1936.

KNOW WELL WHERE YOU SELL

In the purchase and sale of securities, as with anything else, experience and good sense warn you to know your dealer. A reputable broker or investment banker is as important to you in his own field, as a retailer in his.

Too often this wise precaution is taken only as to those from whom you buy. Equally important, and especially in the sale of your own securities, is it important to know him through whom you sell.

If reputable, responsible and trustworthy, your securities dealer will build future confidence based on past performance.

If he is disreputable, irresponsible and untrustworthy, it may cost you your life savings to entrust him with the sale of your securities. Many such dealers depart with your cash or securities and leave no forwarding address.

Look well before you sell. Know well where you sell.—*Boston Better Business Bureau.*

PROTECTION OF CHILDREN FIRST AIM OF FEDERAL CAUSTIC POISON ACT

Fines were recently assessed against a number of manufacturers and distributors, including ten druggists and paint dealers, in the District of Columbia, for selling caustic or corrosive materials in containers which did not meet the labeling requirements of the Federal Caustic Poison Act, according to officials of the Food and Drug Administration, U. S. Department of Agriculture.

Such common household articles as lye, used in softening water, cleaning out drains and kitchen sinks, or ammonia used in cleaning, are dangerous poisons and by law must be labeled as such. Children cannot be expected to read such labels. The products should invariably be stored in a safe place and especially should be kept well out of the reach of children.

Two recent examples of such poisoning have been widely publicized. A small boy in Chicago and another in Washington, D. C., each swallowed lye enough to sear the tissues of the throat and esophagus, until death by starvation appeared certain until proper treatment was instituted. Both are now on the road to recovery.

Congress passed the Federal Caustic Poison Act on March 4, 1927 for the purpose of insuring the use of poison labels on the dangerous products and thus putting users on their guard. Enforcement of the Act was assigned to the Food and Drug Administration.

The law applies to the following substances in the percentages noted, or more: Hydrochloric acid, 10 per cent; sulfuric acid, 10 per cent; nitric acid, 5 per cent; carbolic acid, 5 per cent; oxalic acid, 10 per cent; any salt of oxalic acid, 10 per cent; acetic acid, 20 per cent; hypochlorous acid or its salts, except chlorinated lime to yield available chlorine, 10 per cent; potassium hydroxide, 10 per cent; sodium hydroxide (caustic soda and lye), 10 per cent; silver nitrate, 5 per cent, and ammonia water, 5 per cent.

There are four distinct markings which must appear conspicuously on the label to meet the specifications of the Act. They are as follows:

1. The word "POISON" must be printed in uncondensed Gothic capital letters. These letters must be at least one-third of an inch high if the trade name or any other word on the label contains a letter this large. If there is no letter this large, the "POISON" must not be smaller than the largest letter.

2. Directions for treatment of the poisonous substance must be stated on the label. The Food and Drug Administration has published antidotal treatments for each of the 12 caustic or corrosive substances covered by the act. They are intended primarily to relieve the patient until a physician arrives to administer more thorough treatment, if necessary.

3. The common name of the caustic or corrosive substance must be printed on the label. This not only is informative for those who use it in the home, but it is of particular value to the physician as he can give immediate medical attention.

4. The label also shall bear the name and place of business of the manufacturer, packer, seller, or distributor.

The cases against the ten druggists and paint dealers in the District of Columbia were based on sales of oxalic acid, acetic acid, and ammonia water without the labeling required by the law. The cases were first offenses on the part of the defendants, apparently for this reason, the court, after each had entered a plea of guilty, imposed a nominal fine of $10 in each case. The statute provides for a fine of

not more than $200 or imprisonment for not more than 90 days, or both.—*U. S. Department of Agriculture.*

EDITORIAL NOTE: Whoever notices improperly designated containers should refer the matter to the authorities.

REPORT OF THE DEPARTMENT OF HYGIENE AT HARVARD UNIVERSITY

The first annual report on the newly reorganized Department of Hygiene at Harvard University was issued December 10, 1936, by Dr. Arlie V. Bock, Henry K. Oliver Professor of Hygiene. The report indicated a steady growth of the various types of services rendered to the students and generally improved facilities over those of former years.

The report revealed that 20 per cent of the college students were admitted to Stillman Infirmary during 1935-1936 and that an additional 15 per cent were sent home to bed. A total of 48,843 visits to the various branches of the Hygiene Department were made, but of these only 2,709 resulted in medical excuses from classes being given. This last figure represents "a considerable reduction from the experience of former years".

The 1,463 cases cared for at Stillman Infirmary spent on the average 3.9 days there, while the 211 men who were admitted to hospitals were confined for 12.6 days per man. The majority of the Stillman patients went there for "acute respiratory infections", under the designation of colds. A total of 41 per cent of the hospital cases, 66, were for appendix operations.

The report stated that "more accidents of a serious nature occur in skiing than in any other field of sport. This year a fracture of the femur, two fractures of both bones of the lower leg, and one case of fracture of two vertebrae resulted from skiing". Only two other serious athletic accidents happened during 1935-1936; in hockey and baseball, both being bad fractures of the lower leg. Football men escaped with one serious injury, a fractured clavicle.

The utility of the newly instituted Psychiatric Clinic, begun last year under the direction of Dr. Kenneth J. Tillotson, is found in the 127 men cared for. This department now employs several part-time doctors, one of whom is at the Hygiene office every afternoon and one morning per week. "Ample evidence of the need to expand this work" exists, according to the report, "not because the problem is peculiar to Harvard or any other University, but because general experience of recent years has shown the great frequency with which mental and personality problems, worthy of the attention of a physician or a psychiatrist, are encountered in any community.

"The problems of personality, including maladjustments of all sorts, are outstanding in frequency and present the greatest challenge of the day in the practice of medicine, both in and outside of university circles," Dr. Bock said. He added that "the student himself is often unaware of his difficulties," and "a voluntary approach to problems of this sort is usually essential on the part of the students".

With regard to Stillman Infirmary, the report stated that although many alterations and improvements have been instituted and more will be undertaken as soon as money can be obtained, the infirmary has naturally become antiquated in many respects, since the building has served the university since 1903 and was one of the first institutions of its kind in the country.

The present policy of outside hospitalization in serious cases will be continued, because the scope of hospital practice is beyond the responsibility which should be assumed by the Department of Hygiene, the report said.

Among the many ailments treated were listed nine animal bites, one human bite, and thirteen insect bites.—*University News Office.*

POPULATION TO REACH PEAK IN 1950; THEN DECLINE

A declining national population is inevitable within 15 years unless the birth rate should rise or immigration increase, according to Dr. O. E. Baker of the Bureau of Agricultural Economics.

"The birth rate has declined more than 25 per cent during the last 10 years. If the birth rate continues to decline at this rate," Dr. Baker says, "a maximum population will be reached by 1945 or 1950. Thereafter the population will decline; declining slowly at first, then at an accelerating rate."

A declining national population, with rural surplus and urban deficit in births, will have serious economic and social consequences in Dr. Baker's opinion.

These consequences will develop slowly and silently, he predicts, adding that it probably will be 25 years before many people will realize what is taking place.

"Fifty years from now," Dr. Baker says, "there may be only a third as many children in the Nation as now, and only half as many women of child-bearing age. There will be nearly three times as many old people. Many unemployed urban people will seek shelter and sustenance with relatives and friends on farms. Many of these people will start little farms, and never again return to the cities to live.

"Millions of farm youths will migrate to the cities, many of these migrants will inherit farms, or, through the settlement of estates, acquire mortgages on farms. Wealth — represented by the ownership of land or the income from it — will be transferred to the cities."

Dr. Baker says that other millions of farm youth will begin farming, mostly on farms vacated by the death of aged farmers. Unless these farms are acquired by inheritance, he declares, there will be an increasing number of tenants.

But there is a way to retard these ominous developments, in the opinion of this economist. "They can be retarded," he says, "by a rapid decentralization of population, industry and commerce, by the

spread of part-time farming and by an appreciation by the middle and upper classes of their responsibility for the reproduction of the race and the welfare of the Nation."—*U. S. Department of Agriculture, Office of Information.*

SOCIAL SECURITY, AND PHYSICIANS

Various Social Security Act forms are being distributed at this time to millions of employees and employers.

What is the status of physicians?

1. Old-Age Benefits: A physician who employs one or more persons in his office is an employer as defined by the Social Security Act. He, as well as his employee or employees, is subject to the taxing provisions of Title No. 8 of the Act which deal with *old-age benefits.*

   (a) A physician in the classification of "employer" should fill out the Social Security forms. So should his employee or employees. Forms should be filed with the local postal authorities or the district Internal Revenue Office.

   (b) A physician in the classification of "employee" is considered an independent contractor, and thus is not subject to the taxes imposed on an employee, except where the physician is regularly employed on a full or part-time basis and is receiving a salary.

   The old-age benefit taxes imposed on employers and employees apply to wages paid on or after January 1, 1937. Tax returns must be filed and the tax paid monthly. Information returns must be made quarterly. The present tax is 1 per cent and is imposed on the first $3000 of wages paid to any employee during the calendar year.

2. Unemployment Compensation: Unless a physician has eight or more employees, he is exempt from Title No. 9 of the Act relating to unemployment compensation and is not subject to the payroll tax imposed by that part of the Act.

Detailed information on forms, taxes, procedure, and so forth, will be found in Regulations 91 relating to employees' and employers' taxes under Title No. 8 of the Social Security Act. If a copy of Regulations 91 cannot be obtained from local postmasters, one may be obtained from the Collectors of Internal Revenue of the respective districts.

CORRESPONDENCE

THE NEW ENGLAND REGIONAL COMMITTEE ON FRACTURES OF THE AMERICAN COLLEGE OF SURGEONS

Editor, *New England Journal of Medicine,*

It would appear appropriate to let the readers of *The New England Journal of Medicine* know the personnel and locations of the various Surgeons on The New England Regional Committee on Fractures of the American College of Surgeons. Each of the five states included in this committee has its own chairman, who is responsible for the membership in his own state and the carrying out there of a definite program for the education and improvement in the treatment of fracture cases.

In Boston on September 18, 1936, began the organization of the five New England States into the Regional Fracture Groups of the American College of Surgeons. For some months this has been under way, and approval by the American College of Surgeons has finally been given to all members on this committee from Maine, New Hampshire, Vermont, Massachusetts, and Rhode Island.

The Executive Committee, which lays out the program of activity for each year, consists of the five state chairmen, the four members from New England on the General Fracture Committee of the American College of Surgeons, the Chairman for New England, Joseph H. Shortell, and the Secretary.

The program of activities of this Committee for this year is as follows: (1) completion of membership in the Committee; (2) cooperation with the American Red Cross in its campaign for better treatment of highway accidents; (3) improving the equipment and fracture treatment in all hospitals in the states, and (4) the holding of fracture symposia in the spring of 1937 in each state to bring the subject of fractures to the attention of medical men.

The membership in the Committee stands as approved by the American College of Surgeons as follows:

Maine

State Chairman—Allan Woodcock, Bangor

Harold W. Garcelon, Auburn
William J. O'Connor, Augusta
Roland L. McKay, Augusta
Samuel S. Silsby, Bangor
Peter S. Skinner, Bangor
Raymond E. Weymouth, Bar Harbor
Edwin M. Fuller, Bath
Carl H. Stevens, Belfast
David E. Dolloff, Biddeford
George A. Gregory, Boothbay Harbor
Willard H. Bunker, Calais
Frederick L. Gregory, Caribou
Charles C. Knowlton, Ellsworth
Herrick C. Kimball, Fort Fairfield
Frank H. Jackson, Houlton
Horace L. Gauvreau, Lewiston
Carleton H. Rand, Lewiston
Blinn W. Russell, Lewiston
John F. Hanson, Machias
Joseph B. Drummond, Portland
Charles H. Hunt, Portland
Henry W. Lamb, Portland
Storer W. Boone, Presque Isle
Neil A. Fogg, Rockland
Eugene M. McCarty, Rumford
Stephen A. Cobb, Sanford
George E. Young, Skowhegan
Charles W. Bell, Strong
Edward H. Risley, Waterville
Edward W. Paine, Waterville

New Hampshire

State Chairman—Ezra A. Jones, Manchester

D. J. Sullivan, Manchester
Z. A. Lavoie, Manchester
Robert Graves, Concord
Carleton Metcalf, Concord
Philip McQuesten, Nashua
Timothy Rock, Nashua
Emery Fitch, Claremont
John Gile, Hanover
James Woodman, Franklin
L. A. Middleton, Plymouth
W. H. Lacey, Keene
T. C. Pulsifer, Berlin
Chester Smart, Laconia
G. Harold Shedd, Conway
J. A. Ferguson, Lancester
Henry M. Wiggin, Whitefield
Ralph M. Jones, Whitefield
E. C. Batchelder, Dover
Thomas Luce,* Portsmouth
W. J. Roberts, Rochester
W. J. Dye, Wolfeboro

Vermont

State Chairman—C. P. Chandler, Montpelier

John H. Woodruff, Barre
Frank J. Hurley, Bennington
E. G. Hebb, Bellows Falls
George R. Anderson, Brattleboro
Philip Wheeler, Brattleboro
Robert L. Maynard, Burlington
Benjamin D. Adams, Burlington
George M. Sabin, Burlington
A. C. Kinney, Hardwick
S. S. Eddy, Middlebury
F. W. Harriman, Montpelier
R. H. Bisson, Montpelier
D. J. Sheehan, Newport
Philip A. Goddard, Morrisville
Thomas A. Hack, Proctor
Wilmer W. Angell, Randolph
Charles H. Swift, Rutland
Stewart Ross, Rutland
Edward E. Hinds, Rutland
Leon E. Sample, St. Albans
Paul Bacon, Springfield
Edward H. Ross, St. Johnsbury
W. H. Krause, Windsor

Massachusetts

State Chairman—A. William Reggio, Boston

John D. Adams, Boston
Alexander P. Aitken, Boston
Marshall L. Alling, Lowell
Charles E. Ayers, Milford
A. Leo Brett, Boston
F. Augustus Binford, Hyannis
Francis R. Burke, Quincy
Charles L. Curtis, Salem
George K. Coons, Boston
Frederic J. Cotton, Boston
George D. Cutler, Boston
I. S. F. Dodd, Pittsfield
Joseph H. Fay, Melrose
Arnold P. George, Haverhill
Bernard A. Godvin, Boston
James J. Hepburn, Boston
Otto J. Hermann, Boston
Donald E. Higgins, Hyannis
Benjamin F. Jones, Northampton
Alfred E. Johnson, Jr., Greenfield
Edward A. Knowlton, Holyoke
Harold R. Kurth, Lawrence
Peirce H. Leavitt, Brockton
G. A. Leland, Boston
Henry C. Marble, Boston
Eugene A. McCarthy, Fall River
Gordon M. Morrison, Boston
George W. Morse, Boston
George Mossman, Gardner
Joseph L. Murphy, Taunton
Harvey F. Newhall, Lynn
John W. O'Meara, Worcester
Allan G. Rice, Springfield
Howard W. Rogers, Newburyport
Mark H. Rogers, Boston
James W. Sever, Boston
M. James Shaughnessy, Framingham
Joseph Shortell, Boston
Russell F. Sullivan, Boston
Curtis C. Tripp, New Bedford
Augustus Thorndike, Jr., Boston
Robert Zollinger, Boston

Rhode Island

State Chairman—Dr. Roland Hammond, Providence

Peter P. Chase, Providence
Murray S. Danforth, Providence
G. G. Dupre, Woonsocket
Herbert E. Harris, Providence
John M. Helfrich, Westerly
W. A. Horan, Providence
John P. Jones, Wakefield
S. G. Lenzner, Providence
Henry McCusker, Providence
William A. Stoops, Newport

A one-day clinic (in the morning at the Massachusetts General Hospital and in the afternoon at the Boston City Hospital) was held in Boston on September 19, where illustrated talks and demonstrations of cases were given. Free luncheon was provided at the Massachusetts General Hospital and a dinner attended by 36 men was held in the evening. Sixty-four men registered at the clinics. The following was the program:

Massachusetts General Hospital: 9 a. m.-12 noon.
Epiphyseal Separations—Langdon Parsons, M.D.
Compression Fractures of the Cervical Spine—Sumner Roberts, M.D.
Compression Fractures of the Thoracic Spine—Edwin Cave, M.D.
Question and Answer Period.

*Deceased.

The Prevention of Volkmann's Contractures—Stephen Jones, M.D.

The Routine Treatment of Suspected Head Injuries—John S. Hodgson, M.D.

Fractures of the Shaft of the Humerus—George Van Gorder, M.D.

Böhler Pin and Plaster Treatment of Long Bone Fractures—Richard Dwight, M.D.

Question and Answer Period.

Boston City Hospital: 2-5 p. m.

Dislocation of the Carpal Trapezoid — Thomas Peterson, M.D.

The Nailing Technique in Fractures of the Neck of the Femur—Newton C. Browder, M.D.

Serious Multiple Fractures — Russell Sullivan, M.D.

Fracture Through the Acetabulum — Joseph H. Shortell, M.D.

Ruptures of the Supraspinatus Tendon—Joseph H. Shortell, M.D.

Fractures In and About the Ankle—Frederic J. Cotton, M.D.

Fractures of the Os Calcis — Otto J. Hermann, M.D.

Fractures of the Olecranon — Kenneth Coonse, M.D.

Fractures Through the Epiphyses (end results) —Alexander Aitken, M.D.

AUGUSTUS THORNDIKE, JR., M.D.,
Secretary for New England.

319 Longwood Avenue, Boston.

THE TREATMENT OF VARICES

December 11, 1936.

Editor, *New England Journal of Medicine,*

On reading the article by Drs. Whitney and Consales on the management of patients with varicose veins, in the December 3 issue of the *Journal*, several points came to mind, about which mention should be made. In their summary there appears the statement that "the present-day confusion concerning the treatment of varicose veins is the result of the rapid progress made in the field during the past ten years". The authors are certainly not confused about the treatment, and have written an excellent paper.

They might also have found less confusion in the general literature if they had availed themselves of some of the writings of the past 5 years. The doctors have either not been acquainted with, or have avoided mention of, several excellent articles on the management of patients with varices, as those by Howard, Jackson, and Mahon, *Arch. Surg.*, 1931; De Takats, *Arch. Surg.*, 1933; Faxon, *Arch. Surg.*, 1934, and Edwards, *Surg. Gynec. Obst.*, 1934. Instead of referring to these writers, Drs. Whitney and Consales have referred to Moszkowicz, *Zentralbl. f. Chir.*, 1927, crediting him with the proposal that the saphenous be ligated at the sapheno-femoral junction, in order to avoid recurrences. Actually, Moszkowicz stated that he usually ligated below the sapheno-femoral junction. Furthermore, the main reason that he gave for doing ligations preliminary to injection was that in this way he could prevent embolism and do a better injection by the subcutaneous route. He did make a brief note that the saphenous veins might be ligated at the sapheno-femoral junction, and that this might be better if the vein were dilated high in the thigh. All of us now realize that ligation should not be limited to patients with such a dilation.

The actual classification of the patients for purposes of treatment is very well stated by Drs. Whitney and Consales. It is pleasant indeed that these indications should, in 1936, tally so well with the indications written in the articles quoted above. In particular, the authors have neglected, in describing their treatment of short saphenous varices, to refer to my 1934 article. In this, so far as I know, I was the first to point out that there are essential differences between varices of the short saphenous and varices of the great saphenous. For anatomic reasons, substantiated by clinical evidence, I, at that time, adopted the principle that the short saphenous need not be ligated, no matter how large the varices of that vein were, in distinction to varices of the great saphenous vein.

Yours truly,

EDWARD ALLEN EDWARDS, M.D.

330 Dartmouth Street,
Boston, Mass.

VACCINATION AND INOCULATION SERVICE

December 2, 1936.

Editor, *New England Journal of Medicine,*

I am somewhat in disagreement with the distinguished Vice-President of the Massachusetts Medical Society, regarding the activities of the Department of Health, in regard to preventive inoculations in Boston.

In the toxin-antitoxin inoculations and vaccinations in New York City and other cities that I have studied, the utmost cooperation is provided for physicians, and no one receives these treatments free except the indigent.

In Boston, thanks to the nurses of the Department of Health and school nurses, this type of free service has become quite popular.

The procedure in the toxin-antitoxin campaign is as follows:

Cards are sent out by Dr. Keenan and Dr. Wilinsky to be distributed in schools to the parents of pupils who have not been vaccinated, advising such parents to consult their family doctors. In the parochial schools, a note is appended, that the Department of Health will inoculate the children on a certain date, usually two weeks afterwards. There is no response to this as a rule, and very shortly after the first card is sent out the nurses distribute another card which requests the parents to sign permission for the city to do this work free.

There have been conferences with Dr. Wilinsky by the committee from the Norfolk District Medical

Society, in which Dr. Wilinsky agreed to cooperate, and send out notices to parents of children under school age to have the toxin-antitoxin work done by the family doctor, and to furnish cards to physicians, saying they were authorized to do this work by the Department of Health; the doctors agreeing to do this work for $3.00 for the three inoculations.

Dr. Wilinsky has been quite cooperative vocally, but in action he has been negative. No cards were issued, and the nurses have been quite active in the campaign to have all children inoculated by the Department of Health. I have found that home visits have been made by Department of Health nurses to children under school age, whose parents were perfectly able to pay a doctor, and these same parents have been advised to bring their children to the Well Baby Clinics, for this free service.

Some physicians have complained of this situation to the proper authorities. They were told to bring in names of nurses and patients.

Some years ago, before the present management, I submitted "names" to the Peter Bent Brigham Hospital, about "Hospital Abuse." The hospital did a nice job on me. It made enemies of these patients for life.

I would like to call physicians' attention to the fact that Boston has the largest quota of nurses to the population of any city in the United States, 45 nurses per 100,000 population, while in the City of New York, where cooperation between the health authorities and physicians is marked, there are only 18 nurses per 100,000 population.

At least one-half of these nurses should be transferred to city institutions where they are needed.

The taxpayers should not be asked to bear this excess burden of taxation, to support nurses that are of no help to the community.

Conditions in Boston do not affect Dr. Frothingham, since he is not in general practice and does not suffer from the encroachment of the Department of Health.

Thousands of dollars are taken away yearly from physicians of Boston, by the action of the Department of Health and their nurses in diverting medical service which rightfully belongs to the profession in Boston.

CHARLES MALONE, M.D.

46 St. John Street,
Jamaica Plain, Mass.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn Street, Chicago

December, 1936.

Editor, *New England Journal of Medicine,*

It is very gratifying to be able to tell you that, with the newly enlarged and remodeled headquarters building, the Association has better facilities than ever before for serving your interests and those of the thousands of other members, Fellows, and subscribers to the A. M. A. journals.

Through the Council on Medical Education and Hospitals and the Bureaus of Medical Economics, of Medical Legislation, and of Public Health, the educational and legal standing of physicians is being safeguarded, and the public health benefited in all sections of the country. Other official bodies, including the Councils on Pharmacy and Chemistry, on Foods, and on Physical Therapy, are working diligently to help physicians on problems connected with drugs, foods, and apparatus.

More specifically, the facilities at headquarters make it possible to furnish *The Journal of the American Medical Association* and the eight monthly special journals at a low cost, which in itself tangibly reflects the benefits of professional cooperation and organization.

During the next year, the Association expects to serve you in the most efficient manner possible. We look forward with pleasure to having your cooperation, as in the past. Thanking you heartily,

Very truly yours,

OLIN WEST,
Secretary and General Manager,
American Medical Association.

RECENT DEATHS

CUDDY—JAMES F. CUDDY, M.D., aged 54 years, medical examiner since 1921, died at his home, 585 Main Street, Athol, Thursday, December 3, 1936. He had been a sufferer from heart trouble for several years. Two weeks previous to his death he was taken acutely ill but believed he had recovered and resumed practice the last two days of his incapacitation.

He was graduated from the Baltimore Medical College and had practiced in Athol for 30 years. He was elected last April as vice-president of the Worcester North District Medical Society and had been an unusually active member for a number of years. He was also a member of the Athol Council Knights of Columbus, the Tully Medical Society and of the Athol School Committee.

Dr. Cuddy is survived by his widow, Claire; two sons and a daughter, all of Athol; and by a brother and three sisters.

BUCKLEY—WILLIAM STEPHEN BUCKLEY, M.D., of 253 Market Street, Brighton, Massachusetts, died at his home, December 8, 1936.

Dr. Buckley was born in 1877, graduated from the Harvard Medical School in 1902 and practiced in Brighton for 30 years. During the World War he served as captain in the United States Army and later held the rank of major in the Officers' Reserve Corps.

Dr. Buckley's professional activities also included service with the Boston Floating Hospital, the Carney Hospital and membership on the staff of St. Elizabeth's Hospital.

He was a Fellow of the Massachusetts Medical Society and the American Medical Association.

He was also a member of the American Legion,

Knights of Columbus and St. Columbkille Court, M. C. O. F.

Six sisters, Mrs. M. L. Dolan, Miss Annie, Miss Katherine, Miss Ellen, Miss Mary, and Miss Elizabeth Buckley, and a brother, John Buckley, survive him.

DIERKES—WILLIAM F. X. DIERKES, M.D., of 23 Elm Street, Westfield, Massachusetts, died at Leeds Hospital, December 5, 1936, only eleven days following the decease of his wife.

Dr. Dierkes was born in Philadelphia in 1872. He graduated from the Kansas City University of Physicians and Surgeons in 1920. Previous to studying medicine, Dr. Dierkes had served in the United States regular army under an enlistment in 1893 with Troop D, 8th Cavalry and was discharged in 1896. He again enlisted in the same year and served with Company E, 18th Infantry and was discharged in 1898 because of injuries received in line of duty. During the Spanish-American War he served in the Philippines.

After graduating in medicine he practiced for a short time in Carbondale, Pa., before opening an office in Westfield. He was a Fellow of the Massachusetts Medical Society and the American Medical Association, a member of the Noble Hospital Staff and the local medical society, president of the Gen. Embury P. Clark camp, Vicinity council of United Spanish War Veterans of Massachusetts and assistant deputy inspector of the State Department of Spanish War Veterans.

Dr. Dierkes was especially interested in proctology. An only son, John Dierkes, survives him.

NOTICES

UNITED STATES CIVIL SERVICE EXAMINATIONS

AMENDMENT TO ANNOUNCEMENT No. 1

Junior Medical Officer (Psychiatric Resident), $2,000 a Year
St. Elizabeths Hospital, Department of the Interior, Washington, D. C.

Applications must be on file with the United States Civil Service Commission at Washington, D. C., not later than the following dates:

(a) January 7, 1937, if received from the following States: Arizona, California, Colorado, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, Wyoming.

(b) January 4, 1937, if received from States other than those named in (a), above.

Applications will be accepted for the position of Junior Medical Officer (Psychiatric Resident) from persons now serving an accredited rotating internship. As stated in the announcement, under "Proof of Graduation," the names of persons who are now serving, but who have not yet completed, the required internship will not be certified for appointment until a certificate of the completion of the internship is filed.

Senior Medical Officer—Female (Psychiatry), $4,600 a Year
Junior Medical Officer (Interne), $2,000 a Year
Junior Medical Officer (Psychiatric Resident), $2,000 a Year
St. Elizabeths Hospital, Department of the Interior, Washington, D. C.

Applications must be on file with the United States Civil Service Commission at Washington, D. C., not later than the following dates:

(a) January 7, 1937, if received from the following States: Arizona, California, Colorado, Idaho, Montana, Nevada, New Mexico, Oregon, Utah, Washington, Wyoming.

(b) January 4, 1937, if received from States other than those named in (a), above.

CORRECTION

Dr. Paul W. Butterfield, a co-author with Dr. Olive Gates and Dr. Shields Warren whose article "The Value of Histologic Differentiation of Basal Cell Carcinoma" appeared in the issue of December 3, was incorrectly recorded as a graduate of Tufts College Medical School in "This Week's Issue", on page 1090.

Dr. Butterfield graduated from Boston University School of Medicine in 1934.

REMOVAL

JAMES A. HALSTED, M.D., announces the removal of his office at 264 Beacon Street to the Faulkner Hospital. Telephone Jamaica 3058.

He will continue his office in Dedham which, after February 1, 1937, will be at 743 High Street.

REPORTS AND NOTICES OF MEETINGS

HARVARD MEDICAL SOCIETY

The Harvard Medical Society met on Tuesday evening, October 27, 1936, at the Peter Bent Brigham Hospital, Dr. C. Macfie Campbell presiding. The medical case was presented by Dr. R. Lyons: A 57 year old white male entered the hospital with a history of pain in the chest, and paroxysmal attacks of coughing of 3 years' duration. The pain was more severe in the reclining position, and during the 7 days before entry had increased markedly in intensity. The attacks of coughing had been aggravated by a "cold in the chest", and the patient often experienced sensations of choking and smothering. One year before admission he had received 15 intravenous injections. Physical examination showed irregularity of the pupils, which reacted sluggishly to light. The chest was emphysematous, and there was a large pulsating bulge to the left of the sternum. There was a systolic murmur at the heart apex, and over the pulsating area. The heart rate was 106 beats per minute. There

was increase in breath sounds and bronchial breathing at the left base, and diminished breath sounds, tactile fremitus, and vocal resonance over the left chest anteriorly. D'Espine's sign was present over the fourth thoracic vertebra. There was a pronounced tracheal tug. The blood pressure in the right arm was 135 mm. of mercury systolic, and 90 mm. diastolic. On the left it was 125 mm. systolic, and 80 mm. diastolic. There were no signs of paralysis of the recurrent laryngeal nerve, and the reflexes were normal. The Hinton was positive, the Wassermann negative. The pulse rate was slowed by pressure on the vagus nerve, and by the administration of digitalis. An electrocardiogram showed paroxysmal auricular fibrillation. X-ray examination of the chest revealed a large mass in the left upper chest, with questionable erosion of the vertebral bodies. The patient was given mercury by mouth and by intramuscular injections, with some relief of his pain.

Dr. Marshall Fulton remarked that it was unusual for an aneurysm of the aorta to reach the size of the mass observed in this patient without more erosion of the bone occurring. He raised the question of whether the mass might be a collection of pulsating fluid without the presence of an aneurysm. Dr. Merrill Sosman commented on the x-ray findings, stating that the shadow of the mass in the postero-anterior view was indistinct, while aneurysm shadows are usually very clear cut. Pulsations were not marked. He believed that this was an instance in which direct inspection was of more value in making the diagnosis of aneurysm than the refinements of x-ray technic.

Dr. William I. Abel presented the surgical case: A 56 year old white housewife had led a perfectly normal physical and mental existence until 2 weeks before entry, at which time she suffered the onset of acute pain over the left eye, and motor aphasia. These conditions progressed in severity until she was unable to communicate with her family, although she could read and write, and apparently understand conversation. On entry she was semi-stuporous, and unable to answer questions, although the physical examination was negative except for a blood pressure of 150 mm. of mercury systolic, and 100 mm. diastolic. Suspicion of toxic psychosis from drugs as the etiologic factor was considered. Ophthalmoscopic examination showed papilledema of the left optic disc, and slight edema of the nasal portion of the right optic disc. Lumbar puncture showed a pressure of 160 mm. of water, with normal dynamics. Spinal fluid proteins were normal. X-ray examination revealed cloudiness of the right frontal sinus, indicative of long-standing sinusitis. Her condition became progressively more lethargic, and the spinal fluid pressure became elevated to 260 mm. of water. Both optic discs became completely choked. The neurologic examination was negative objectively at all times. The white blood count varied between 7000 and 9000.

Dr. Elliott C. Cutler in discussing the case demonstrated the inability of the patient to name objects, although she still could articulate well. He stated that ventriculography and encephalography were contraindicated because of the danger of breaking down the barrier of resistance in case the patient had a cerebral abscess. The lesion was believed to be very localized because of the absence of neurologic signs, and the consensus of the surgical staff was that it was neoplastic in type. Dr. Macfie Campbell emphasized the importance of the finding that the patient could articulate well, and could understand conversation. He believed that the sensory form of aphasia as exhibited in this case made the accurate localization of the lesion difficult. The extreme degree of papilledema was considered an indication for immediate operation.

Dr. Merrill Moore, Associate in Psychiatry in the Harvard Medical School, presented a paper on "Cases of Attempted Suicide in a General Hospital." Dr. Moore emphasized the importance of suicide as a cause of death, since annually it is responsible for more deaths than duodenal ulcer, poliomyelitis, leukemia, Hodgkin's disease, and many other medical causes of death combined and especially since it may be a preventable cause of death. A survey of 1147 cases of attempted suicide admitted to the Boston City Hospital between the years 1915 and 1936 revealed a steady increase in the number of cases each year. Thus in 1915 there were but 24 cases, while in 1935 there were 99. Six hundred and sixteen of these cases were females, while 531 were males, figures corresponding to the sex distribution of the general population of Greater Boston. Eleven per cent of the attempts were successful, and resulted in death before or after admission to the hospital. Only 9 per cent of the females were successful as compared with 13 per cent of successful males, the explanation of the greater success among males probably being due to the more aggressive personality of men, their mechanical ability and greater knowledge of chemicals in taking poison by mouth. Although the majority of cases were treated on the medical wards, Dr. Moore pointed out that most of the cases actually presented problems in social or psychologic medicine. Seven per cent of the total number were sent to mental hospitals for observation and treatment.

Sixty-one per cent of the females were between the ages of 16 and 30 years, and the majority of these were between 21 and 25 years. There was a definite decline in cases attempting suicide among females after the menopause. This may in some way be correlated with the individual's sex drives. Only 33 per cent of the males attempted suicide between the ages of 16 and 30, indicating that for males suicidal impulses are more sustained and continue into later years.

A study of the causes for the attempted suicides showed that difficulties in connection with jobs were most important among males, while domestic troubles were given the most common reasons by women. Third in ranking reason was ill health, while the love motive was of least importance, according to this series. The reasons for the seeming

unimportance of love troubles may be due to reticence on the part of the subject to mention such difficulties.

Neither drug addiction nor alcoholism was of much importance as a cause for attempted suicide. The methods most frequently used were taking poison by mouth, or inhalation of illuminating gas. *Of 322 patients taking iodine not one died from the effects of this poison.*

Among many interesting facts revealed by this study were the findings that very few people attempted suicide shortly after eating or "on a full stomach". Among women most attempts were made on Wednesday, while men were most prone to choose Sunday for their efforts. There were definite increases in the number of suicides among women in the months of May, July, August and September, increases which may be connected with some vestigial sexual relation between these months and primitive mating seasons. More males attempted suicide in April.

Dr. Moore emphasized the fact that the figures which he presented were not representative of the community as a whole, but only of the Boston City Hospital. A survey of the cases seen by the medical examiner would undoubtedly reveal very different findings.

He deplored the existing treatment of cases of attempted suicide, since they are discharged after routine treatment on regular medical and surgical wards without investigation of the causes and conditions leading to the attempt. The subject of suicide is often ignored in the home of the individual because of the social stigma connected with the act. Dr. Moore advocated the establishment of "protective wards" in which cases of attempted suicide could be given a thorough examination, where a psychiatric examination could be done and where the benefits of the social service department could be made available. Patients from such a ward could be followed in a psychiatric clinic of the outpatient department. Such a method would enable a scientific approach to the problem of the individual and social forces, leading to suicide.

Dr. Macfie Campbell, in discussing Dr. Moore's paper, commented on the great number of cases of attempted suicide which were *not* successful. Such attempts are often not pressed very hard, and are frequently made in the presence of an audience. He raised the question of whether a man has the right to commit suicide, and pointed out the striking difference in racial beliefs on this subject. He remarked that it was really remarkable that more people did not commit suicide when the miserable conditions in which a large portion of the earth's population is forced to live is taken into consideration.

THE CONNECTICUT SOCIETY OF PSYCHIATRY

The quarterly meeting of the Connecticut Society of Psychiatry was held at Stamford Hall in the afternoon on December 3, 1936. A paper was presented by Dr. Karl M. Bowman, Director of the Psychiatric Division of Bellevue Hospital in New York City, treating with the present trends in hospitalization for mental diseases in both general and special hospitals. A discussion followed in which Professor Kahn of Yale University, Dr. Roy L. Leak, Superintendent of Middletown State Hospital, and Dr. Chester Waterman of Norwich State Hospital, participated. The meeting was opened by the President, Dr. Otto G. Wiedman of Hartford, and the speaker was introduced by Dr. Francis M. Shockley, Physician-in-Charge of Stamford Hall.

After the meeting, dinner was served and a social hour followed.

WACHUSETT MEDICAL IMPROVEMENT SOCIETY

The third lecture in the course in "Parliamentary Law for Physicians" will be given at Holden District Hospital, Tuesday, December 22, 1936, at 8 p. m., by Charles W. Proctor, Esq.

The topic will be "General Parliamentary Procedures." All physicians and nurses are invited to attend.

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

The Worcester North District Medical Society will hold a regular quarterly meeting at the Leominster Hospital, at Leominster, at 4:30 p. m., Wednesday, January 27, 1937.

Dr. L. E. Phaneuf of Boston will speak on "The Significance of Menorrhagia and Metrorrhagia," with illustrations. Dinner will be served at 6 p. m.

F. M. McMURRAY, M.D., *Secretary.*

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

A stated meeting of the Essex South District Medical Society was held at the Salem Hospital, December 2, 1936.

At 5 p. m., members of the Salem Hospital Staff presented a clinic, covering nine different subjects of medicine or surgery. The clinic was very interesting and instructive.

After a dinner at 7 p. m., the guest speaker, Dr. John W. Strieder, of the Boston University School of Medicine, presented the subject of "Pulmonary Suppuration."

His illuminating presentation was supplemented by lantern slides and his subject was made practical and instructive.

NATHANIEL POPE BREED, M.D., *Reporter.*

The regular meeting of the Essex South District Medical Society will be held Wednesday, January 6, 1937, at the Danvers State Hospital, Hathorne.

Clinic at 5 p. m., dinner at 7 p. m.

The very interesting talking picture, tracing the growth and development of the human infant, en-

titled "Life Begins," by Dr. Arnold Gesell, of Yale University Clinic of Child Development, will be shown at approximately 8 p. m.

R. E. STONE, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, DECEMBER 21, 1936

Monday, December 21—

8:15 p. m. Boston Medical History Club. Boston Medical Library. 8 Fenway.

Tuesday, December 22—

11:30 a. m. Massachusetts General Hospital. Eye Nerve Conference. Outpatient Department.

Wednesday, December 23—

†12 m. Clinical-Pathologic Conference. Children's Hospital Amphitheater.

2 p. m. Massachusetts General Hospital. Psychiatric Clinic. Outpatient Department.

4 p. m. - 5 p. m. Surgical Pathological Conference. Dr. Cutler and Dr. Wolbach, Peter Bent Brigham Hospital.

Thursday, December 24—

*8:30 - 9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and the Children's Hospitals, held this week at the Peter Bent Brigham Hospital.

9 a. m. Massachusetts General Hospital. Surgical Grand Rounds.

9:15 a. m. Massachusetts General Hospital. Neurological Conference. Ether Dome.

12 m. Massachusetts General Hospital. Clinical-Pathologic Conference.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

December 17—Medical Clinic at the Peter Bent Brigham Hospital at 3:30 p. m. by Dr. William P. Murphy.

December 17—Massachusetts General Hospital, Clinical Meeting of the Staff at 8:15 p. m. in the Moseley Memorial Building.

December 17—Boston Society of Psychiatry and Neurology. See page 1145, issue of December 10.

December 21—Boston Medical History Club. See page 1145, issue of December 10.

December 22—Wachusett Medical Improvement Society. See page 1197.

January 10 - March 21—Sunday Afternoon Lectures at the Harvard Medical School. See page 1141, issue of December 10.

January 14—Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

January 15—William Harvey Society, 8 p. m. in the Auditorium of the Beth Israel Hospital, Boston.

January 15—Boston Society for the Advancement of Gastroenterology. See page 1145, issue of December 10.

February 3—American Social Hygiene Association. See page 1186.

February 25, 26, 27—The New England Hospital Association. Hotel Statler, Boston.

March 30 - April 2—First International Conference on Fever Therapy. Postponement notice. See page 52, issue of July 2.

April 21-24—American Society for Experimental Pathology. See page 1075, issue of May 21.

October 25-29—American College of Surgeons. Chicago, Illinois.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

January 7—See page 1197.

FRANKLIN DISTRICT MEDICAL SOCIETY

Will meet at the Weldon in Greenfield at 11 a. m. the second Tuesdays of January, March and May.

CHARLES MOLINE, M.D., Secretary.

Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

January 13—Bear Hill Golf Club, Stoneham.
March 16—Danvers State Hospital, Danvers.
May 11—Bear Hill Golf Club, Stoneham.

KENNETH L. MACLACHLAN, M.D., Secretary.

1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

January 19—8:15 p. m. The Peter Bent Brigham Hospital. Communications and Case Presentations by the Staff. Suggested title—"Abdominal Pain from the Medical and Surgical Standpoint." Details of program to be announced.

February 23—Time, place and details of program to be announced.

March 30—8:15 p. m. New England Deaconess Hospital. A Symposium on Diabetes entitled "A Survey of the Diabetic Work of the George F. Baker Clinic in the New England Deaconess Hospital." Communications and Case Presentations by the Staff. Drs. Elliott P. Joslin, Howard F. Root, Priscilla White, Alexander Marble and Allen P. Joslin.

May—Annual Meeting. Details to be announced.

Note: The Censors will meet for the examination of candidates on the first Thursday of May, 1937. Fee of $10.00 is payable at the time of examination. Application blanks may be obtained by writing the Secretary, furnishing name, address and name of school of graduation in medicine. Application must be made at least three weeks prior to date of examination. Candidates whose applications are on file will receive proper notices.

FRANK S. CRUICKSHANK, M.D., Secretary.

1247 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

January 21—11 a. m. Bridgewater State Farm.
March 18—11 a. m. Brockton Hospital.
April 15—Annual Meeting. 11 a. m. Ducy Hospital.
May 20—11 a. m. Lakeville State Sanatorium.

FRED F. WEINER, M.D., Secretary.

231 Main Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

January 27—Boston Medical Library. 8:15 p. m. Joint Meeting with the Boston Medical Library. "Anthropology." Dr. Carleton S. Coon.

March 31—Boston Medical Library. 8:15 p. m. "Social Insurance—It Affects the Medical Profession." Dr. Charles E. Mongan. Discussion: Dr. Channing Frothingham.

April 28—Annual Meeting. Boston Medical Library. 8:15 p. m. "Problems in Surgical Diagnosis." Dr. Howard M. Clute.

CONRAD WESSELHOEFT, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

January 13—Worcester City Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

February 10—Worcester State Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

March 10—The Memorial Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

April 14—Worcester Hahnemann Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

May 6—At 4:30 in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the spring meeting of the Board of Censors.

Wednesday Afternoon and Evening, May 12—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.

27 Elm Street, Worcester.

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

January 27—See page 1197.

BOOKS RECEIVED FOR REVIEW

Allergic Diseases. Their Diagnosis and Treatment. Ray M. Balyeat, assisted by Ralph Bowen. Fourth Edition, Revised and Enlarged. 516 pp. Philadelphia: F. A. Davis Company. $6.00.

Modern Treatment and Formulary. Edward A. Mullen. 707 pp. Philadelphia: F. A. Davis Company. $5.00.